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CERVICAL-CORD INJURIES*

A Study of 101 Cases

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EVERY discussion of any level of spinal-cord injury should be based on two fundamental concepts. These are that spinal-cord injuries are different from spinal-column injuries without cord involvement, and that all significant spinal-cord injuries are accompanied by spinal shock and its effects. One has only to refer to the literature to discover many authors who draw conclusions concerning the diagnosis, treatment and prognosis of a neurologic injury from a mixed series of both cord and vertebral injury cases. Wegner and I[1] drew attention to this in 1939 in relation to a study we made at that time of the bone lesions accompanying cervical spinal-cord injuries. When a spinal-cord injury is present, any associated bone injury is secondary and of relatively little importance. Under such circumstances, diagnosis, therapy and prognosis are all primarily problems of the cord injury, and the bone damage is from every point of view subordinate. Only when there is a back or neck injury in which the spinal cord has been undamaged do the bony structures and any changes in them properly assume first place in the consideration of the case. Until this is generally recognized and acted on but little improvement can be expected in the functional end results of spinal-cord injuries.

This report deals only with acute injuries to that part of the spinal cord that is enclosed within the seven cervical vertebras. As a necessary part of the study, however, consideration has also been given to any associated bony and ligamentous damage done to the cervical spine. There are 101 cases in this series. Fifty-five patients survived, and 47 of these have been followed to an end result for periods varying from six months to fifteen years. Forty-six died, a mortality of 46 per cent. Autopsies were obtained and the cords studied in 17 cases.

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Spinal Shock

Spinal shock is a constant accompaniment of significant spinal-cord injuries. It is recognized by its effects and is known to occur at once after the infliction of the injury and to last for periods varying from a few hours to months. It has nothing to do with surgical shock. As the result of its presence, all reflex activity below the level of the injury becomes chaotic, unpredictably variable and devoid of any diagnostic or prognostic significance. The deleterious effect of spinal shock is greatest on those reflexes mediated by the spinal segments immediately below the level of injury and decreases proportionately as lower segments are reached. As the location of the injury descends, bladder difficulties and bedsores, for example, become increasingly common because of the closer approximation of the segments controlling these reflexes to the level of injury.

Nature of the Accident

The commonest cause of a cervical-cord injury in this series was by all odds a fall, usually on stairs. Fifty or nearly half of the patients were injured by a fall, in 34 cases on stairs (Table 1). Twenty-four of them died. The next commonest cause was diving, with 8 cases. Only 2 of these patients died. It was not possible to learn the nature of the accident in 16 cases, in 12 of which the patients died. Alcohol is evidently a factor in the production of this kind of injury. Acute alcoholism was present in 23 cases and was almost three times as common in the fatal accidents (17 cases) as in the nonfatal (6 cases). In 15 cases it was associated with the fall that caused the injury. In the other 8 cases the kind of accident could not be determined because of its presence.

Bony and Ligamentous Injuries

Although bony and ligamentous injuries are of less importance than cord injuries, it is more convenient to deal with them first (Table 2). It is apparent that the commonest injury to the cervical spine is one that involves multiple vertebras. An analysis of the 36 cases of multiple injury shows that 26 centered about the fifth vertebra and 2 others included it. Nineteen patients recovered and 17 died. More than half these injuries caused a dislocation as well as a fracture determined it proved to be centered around the fifth and sixth cervical vertebras, with the greater number involving the fifth.

Forty-four cases with bone injury had an associated dislocation and in all but 1 case the dislocation was anterior (Fig. 1). It is probable that with the exception of the first and second vertebras a true posterior dislocation of the cervical vertebras (Fig. 2) almost never occurs, and then only rarely in an acute form. This group includes one such case. The patient was struck on the forehead, his head was driven backward, the odontoid was broken, and a posterior dislocation of the skull and atlas on the axis was produced. Fortunately the dislocation was not lethal. Despite its rarity a diagnosis of posterior dislocation is frequently made by x-ray. In this case the mobilized part of the column moved backward in relation to the immobilized part. In the common anterior dislocation the mobilized part of the column moves forward in relation to the immobilized part. If all other factors are omitted, the presence of a dislocation in addition to a fracture increases the chance of death slightly, the mortality being 52 per cent in this group as opposed to 44 per cent in the non-dislocation group.

The ligamentous cases deserve special emphasis. It has been shown that it is possible and must be fairly common for adjacent cervical vertebras to be dislocated either partially or completely without bone injury and by virtue of a tearing of the capsular and other ligaments that surround the articular facets.[1] This series includes 11 such patients, none of whom had any demonstrable injury of the vertebral column. The cord injuries included among other diagnoses classic cervical hematomyelia and transection. This has convinced me that it is possible for cervical vertebras to be momentarily dislocated sufficiently far to damage the cervical cord, even to the point

of the involved vertebras. In the single fractures, whether with or without dislocation, the same preponderance of location appears, 21 involving the fifth and 18 the sixth vertebra. Moreover, in the one patient who had a rupture of a cervical intervertebral disk with an immediate and acute extrusion of the nucleus pulposus, the involved disk was that which separated the fourth and fifth vertebras. Thus in 60 of the 93 patients in whom the site of the bone injury was certainly

TABLE 1. *Causes of Cervical-Cord Injury.*

NATURE OF ACCIDENT	TOTAL PATIENTS	LIVING PATIENTS	DEATHS	PATIENTS WITH ASSOCIATED CEREBRO-SPINAL INJURY
Fall:				
On stairs	34	17	17	6
Otherwise	16	9	7	2
Diving accident	8	6	2	1
Automobile accident	9	6	3	2
Struck on head	5	4	1	2
Stab wound	2	2	0	0
Machine-shop injury	2	0	2	1
Struck by streetcar	2	1	1	0
Struck by train	1	0	1	1
Athletic injury:				
Football	1	1	0	1
Wrestling	1	1	0	0
Ice hockey	1	1	0	1
Accidental burial	1	1	0	0
Assault	1	1	0	0
Recurrent dislocation	1	1	0	0
Cause unknown	16	4	12	2
Associated alcoholism	23	6	17	

TABLE 2. *Location of Injury and Outcome.*

DIAGNOSIS	OUTCOME OF CASE	INVOLVED VERTEBRAS									X-RAY EXAMINATION		TOTAL
		1	2	3	4	5	6	7	MULTIPLE	UNDETERMINED	NONE	NEGATIVE FOR FRACTURE	
Ligamentous injury with dislocation	Living						4	2		5		11	11
	Dead						0	0		0			0
Fracture with dislocation	Living	1			0	4	4	1	11				21
	Dead	0			3	6	3	1	10				23
Fracture only	Living			1		7	2	1	8				19
	Dead			1	1	2	5	0	7				16
Stab wound	Living					2							2
	Dead					0							0
Acute rupture of intervertebral disk	Living				1								1
	Dead				0								0
Type of injury undetermined	Living										1		1
	Dead										7		7
Totals		1	0	2	5	21	18	5	36	5	8	11	101

of transection, only to return to apparent normality long before the physician sees the patient. None of these 11 patients died. The location of the injury as evidenced by the segmental level of the symptoms was again concentrated around the

FIGURE 1. *Anterior Dislocation of Cervical Spine (lateral view).*

fifth cervical vertebra, with a tendency to move downward, particularly in the transected cases. As has been pointed out,[1] it is these injuries that are apt not to unite until late, because healing depends solely on ligamentous repair.

One should not jump to the conclusion from these figures that the preponderance of acute cervical vertebral injuries involve the fifth vertebra. Indeed, it can be easily demonstrated that they do not. The present figures apply only to the patients with acute cervical cord injuries. A realization that any significant acute injury to the cervical cord at or above the fourth cervical segment produces complete respiratory paralysis will explain why cases of damage to the higher cervical vertebras were so rare in this series. Either the patient was dead before arrival at the hospital and was taken directly to the morgue or was not suffering from involvement of the cord as the result of the accident.

Pathology of Cord Injuries

The pathologic changes produced in the cord by the injury were confirmed by autopsy, operation or sufficient clinical observation to be unmistakable in 68 of the 101 cases.

There were 13 transections. All the patients died except 2, who were living six months and three years, respectively, after the accident. In one case the transection developed ten days after injury. In this interval a diagnosis of mild contusion had been made and was thought to have been confirmed by operation. The transection developed suddenly and is believed to have been

FIGURE 2. *Posterior Dislocation of Lumbar Spine (lateral view).*

caused by interference with the cord circulation locally, resulting in something of the nature of a thrombomyelitis. Respiration was entirely diaphragmatic, and what little motion remained in the arms and shoulders was entirely useless. Of the remaining 11 cases the diagnosis was confirmed clinically in 2, by operation in 1, and by autopsy in 8. In all, the damage lay between the fifth and eighth cervical segments.

There were 6 contusions of the cord. Two were verified by operation, 2 by clinical observation,

and 2 at autopsy. Only 1 patient survived. Six cases originally diagnosed clinically as contusion proved to have transections at autopsy (5 cases) or at operation (1 case). No patient originally diagnosed as having contusion was discharged with that diagnosis. There were 6 cases with pure compression of the cord, without other myelogenous damage. Two patients survived, with clinical confirmation in one case and operative confirmation in the other. In the 4 fatal cases, the diagnosis was confirmed by operation in 2 and by autopsy in 2. The presence of edema of the cord was never confirmed by either operation or autopsy. There were 5 cases in which the clinical findings justified this diagnosis. All except one of the patients survived.

The commonest pathologic condition was hematomyelia, with or without edema. It is possible that some of the surviving cases classed as hematomyelia actually had a mild contusion. Short of microscopic examination such a differentiation is impossible. Because the symptoms and course were analogous to those of proved cases of hematomyelia, however, they have been classified in the latter group. There were 35 of these cases. Twenty-nine were confirmed clinically, 3 by operation, and 3 by autopsy. Twenty-six patients survived.

Two cases of stab wounds and 1 of radiculitis were confirmed clinically, and all the patients survived. In 3 cases the diagnosis was not certainly known and in 30 it could not be verified. Because the cord lesion has necessarily to be located below the fourth segment if a patient with any significant acute cervical-cord injury is to live long enough to be hospitalized before he dies of respiratory failure, and because the area of cervical cord below this point is so small that an estimate of the differential effects of injury at different segmental levels within this region is impossible, no effort has been made to study the distribution of the cord injury in more detail.

Diagnosis

It seems certain that the best one can do in making a diagnosis at or close to the time of the sustaining of a cord injury is to determine the approximate upper level of the damage by a sensory examination. Except for this information the doctor is limited to the decision whether the lesion is of the type that causes a block of the flow of the cerebrospinal fluid. The lesions that cause such a block are edema, with or without hematomyelia, contusion, compression and concussion of the cord. The compression may be caused by a meningeal clot, a fragment of fractured bone, the crushing of a vertebral body, a dislocation of a vertebra or an extrusion of a nucleus pulposus. Contusion is self-explanatory and may be combined with compression. Concussion is associated only with bullet wounds of the bony wall of the canal. The lesions that do not cause a block of the flow of the cerebrospinal fluid are hematomyelia without edema, anatomical transection and rarely an overlapping tear in the dura and arachnoid with an undetermined amount of cord injury. This last permits cerebrospinal fluid to be diverted to the extradural or subdural space and thus prevents filling of the subarachnoid space below the point of injury. Such findings may lead to the diagnosis of cerebrospinal-fluid block when none is actually present. Conditions that produce no block may be present as a complication of any of those that do cause a block and, of course, be unrecognizable, but the reverse is never true. Final differentiation between pathologic entities within these two classifications must await the final outcome of the case and may not be possible for months or even years. Even then it may be impossible unless major sepsis is not present, the patient is not suffering from hypoproteinemia, the bladder has reached a satisfactory functional end point, and proper physiotherapy and splinting have been practiced.

Disagreement between the immediate and final diagnoses is common in all cord injuries. Even observations made of the exposed cord at operation are not necessarily accurate or dependable. This is particularly true when the cord looks as though it had sustained little or no damage so far as its surface goes. Because the vascular supply to the cord is minimal and so constructed as to produce a bottleneck[2, 3] in both the arterial and venous channels, its tissue is particularly vulnerable should damage to the arteries or venous thrombosis occur. Cells that would not have sustained lethal damage in any other tissues do not recover, and a spreading myelomalacia may develop in the presence of damage to a single artery or of thrombosis of a single vein at certain levels on account of the lack of collateral circulation. A detailed study of this series has demonstrated that in 32 cases the immediate and final diagnoses agreed and in 36 they did not agree. Whether there was agreement or disagreement could not be determined in 33 cases (Table 3). Agreement and disagreement were determined by autopsy, by operation provided the patient lived more than forty-eight hours or by clinical observation provided he lived one year or longer after the accident. The immediate diagnosis was that made as soon after the accident as adequate studies had been completed. Under these conditions agreement was held to be present in 3 cases of transection, 22 cases of hematomyelia, 3 cases of compression without other pathology, 1 case of edema, 2 cases of stab wounds and 1 case of radiculitis.

A preliminary diagnosis of contusion of the cord was never verified. Thus, only 28 of 54 cases were correctly diagnosed when first seen. Only 1 of the 11 diagnoses made at operation was confirmed later. This was in a case of compression. Of those cases in which the two diagnoses did not agree the patient's condition was worse at discharge than it was at admission in 26 cases.

In summary, it seems reasonable to conclude that a diagnosis of hematomyelia is the most apt to be correct, and if wrong is least liable to turn out to be something more serious. On the other hand, contusion, compression and edema are the conditions least apt to be correctly diagnosed and the most likely to be finally diagnosed as a condition that is much more serious than the original one was thought to be. This last is true not so much because of inaccuracies at the time the first observations are made as because of the progressive disintegration in the cord tissue that goes with compression of the cord. This compression may come from within and be traceable to edema, large hemorrhages or the results of altered circulation, or from without because of the effect of bone fragments, meningeal clots and the like. This fact has its practical aspect in that it points to the desirability of relieving extraneous pressure as early as possible, and at the same time emphasizes the danger attendant on operative procedures that are carried out in the face of a cord already damaged by edema or by hemorrhage within its substance. In particular it indicates the folly of splitting the cord and thus running the risk of producing damage that might not otherwise occur.

Diagnostic and prognostic conclusions based on the onset, type and extent of motor and reflex disability are never justified. Recognition of this fact is extremely important. It comes about because of the changes wrought by spinal shock. Fifty-five of the patients in this series could be studied in detail from this point of view (Table 4).

In the light of this evidence it cannot any longer be seriously contended that either the rapidity of onset or the amount of paralysis following an injury to the cervical levels of the spinal cord is of any diagnostic or prognostic significance whatsoever.

The demonstration of the presence or absence of a block of the flow of cerebrospinal fluid is made by a study of the total protein content of the fluid below the block and the performance of a fractional Queckenstedt test. Evidence of block consisting in an increase in total protein frequently appears before the evidence of dynamic block, and always outlasts it. The fractional Queckenstedt test is best done by compressing the jugular veins, not by hand but by inflation and deflation of a blood-pressure cuff wrapped around the patient's neck. Observations on the intraspinal pressure are made and recorded with every 10 mm. increase of pressure in the cuff from 0 to 40 and similarly with every 10 mm. decrease from 40 to 0. Partial dynamic blocks are recognizable by this method when every other has failed. As has been pointed out above, if a block is present, the patient is suffering from either edema, with or without hematomyelia, contusion, compression or concussion of the cord. If a block is absent, he is suffering from either hematomyelia without edema or an anatomical transection. The only source of diag-

TABLE 3. *Difference between Immediate and Final Diagnoses.*

Immediate Diagnosis	Total No. of Cases	Diagnosis Confirmed	Diagnosis Altered					Final Diagnosis		
			Transection	Contusion	Compression	Edema	Hematomyelia	Living Patients	Deaths	Total No. of Cases
Transection	4	3					1	2	11	13
Contusion	10	0	6		2		2	1	5	6
Compression	14	3	3	3			5	2	4	6
Edema	8	1	1		1		5	5	0	5
Hematomyelia	28	22		2		4		26	9	35
Stab wound	2	2						2	0	2
Radiculitis	1	1						1	0	1
Fractured spine	1	0		1				0	0	0
Totals	68	32	10	6	3	4	13	39	29	68
Not made										33
Grand total										101

TABLE 4. *Onset and Type of Paralysis.*

Diagnosis	Total No. of Cases	Onset		Type	
		Immediate	Delayed	Complete	Partial
Transection	10	6	4	3	3
Contusion	5	1	4	0	1
Compression	5	2	3	2	0
Edema	4	0	4		
Hematomyelia	27	17	10	8	9
Stab wound	2	1	1	0	1
Radiculitis	2	1	1	0	1
Totals	55	28	27	13	15

nostic error lies in the rare possibility of the patient's having coincidental tears in the dura and arachnoid and presenting signs that superficially appear to indicate a block when there is actually none present. Further diagnostic differentiation is unjustified and impossible until the lesion has reached an end point.

Nonoperative Treatment

Certain general principles of treatment must be adhered to if the end results that the patient has a right to expect are to be obtained. In every case of spinal-cord injury the bladder should be emptied and an inlying catheter fastened in place at the earliest possible moment. As soon afterward as it can be done this catheter should be attached to a tidal-drainage apparatus properly adjusted by cystometry to the needs of the bladder. Simultaneously the patient should be treated for surgical shock if and when it is present. Both steps should precede all but the most superficial diagnostic measures. The demonstration of the presence or absence of a block should always follow the above and come before all other measures. Splints, no matter what their material, that are fixed in relation to the patient must never be used in spinal-cord injuries, especially in thoracic and lumbosacral ones. In particular the application of plaster of Paris is contraindicated regardless of the amount of padding used. So, too, are rubber and other rings or doughnuts, Bradford or other frames and water or air mattresses. Metal frames and any other method that aims to accomplish rapid hyperextension are also contraindicated. Sufficient protein must be given in the diet or by transfusions or other means to maintain the serum protein at 6.0 gm. per 100 cc. or better. The salt intake must usually be increased also, and the patient should be started at once on large amounts of all the vitamins. While he remains on tidal drainage the fluid intake should never be allowed to fall below 4000 cc. per twenty-four hours for an adult with a normal cardiorenal system. Care must be taken to keep the bowels open and prevent the development of fecal impactions. All patients must be moved off the back and onto one or the other side every two hours, day and night, during their stay in bed. They should never be allowed to lie in a wet bed for even as little time as fifteen minutes. These are general measures that apply to all patients with spinal-cord injuries until such time as anatomical and functional end results have been reached, regardless of the diagnosis. Other procedures depend on the presence or absence of a positive Queckenstedt test. This reaction must be determined in every case without exception, and the test should be made after the patient is out of surgical shock and on tidal drainage.

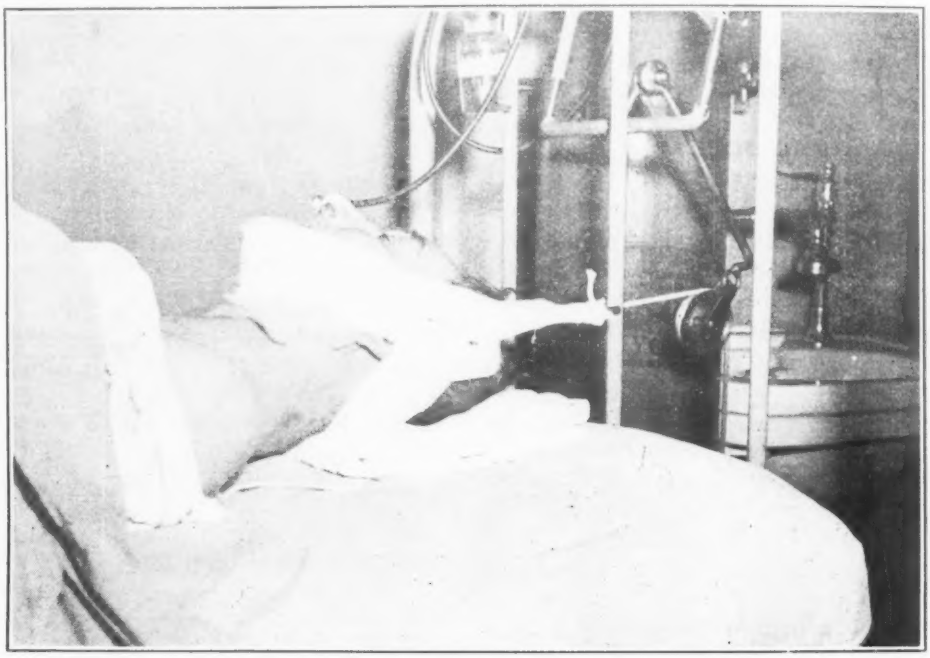

FIGURE 3. *Bridle Made from Folded Flannel Bandages.*

It is vitally important in all patients with cervical-cord injuries that the head be not accidentally flexed. This control of the movements of the head on the spine is equally essential whether or not there is a cerebrospinal fluid block. It is

best accomplished by traction of the head in extension with a bridle. It can be achieved by the application of some form of fixed splint, such as a Thomas collar or a plaster cuirasse, with or without a jury mast, but my experience has shown that these applications can do no more than traction does, usually do not do so much, and are much more difficult and dangerous to use. Under the circumstances the best apparatus is the one that is simplest and easiest to apply. As can be seen in Figure 3, such an apparatus can be made from two flannel bandages each folded lengthwise a number of times until an appropriate length is reached. One of these bandages is looped around the chin and its free end attached to a spreader, which in its turn has a piece of rope hung from its center point. This rope passes over the pulley of an ordinary Buck's extension apparatus that is attached to the bed, and terminates in a loop that supports a 5-pound weight. The other bandage is looped about the occiput and the free ends are pinned to those parts of the first bandage that are opposite the ears. The head of the bed should be slightly elevated. By adjusting the relative lengths of the bandage loops and the height of the pulley in relation to the line of pull, traction can be exerted on the head with simultaneous control of the flexion and extension of the neck. Commercial bridles have no advantage and offer many disadvantages compared with this simpler one.

In general and unless there is some special indication, traction in all cases of cervical-cord injury should be parallel to the long axis of that part of the cervical spine that has not been dislocated or moved. Under such circumstances it is well to put a soft pillow under the head. If extension is required, a small hard pillow should be put under the shoulders. I have not used tongs but have used piano wire looped through adjoining trephines on either side of the vertex of the skull as a means of applying traction and using greater weights than are possible with the flannel-bandage bridle. I have been unable to accomplish anything more by this method than I was able to do with the bridle. Regardless of the weight applied, all that traction can do is to stretch the muscles and ligaments of the neck and tire out the former. The constant application of 5 pounds of traction will accomplish this as expeditiously and as efficiently as will a heavier weight. Neither method nor any amount of weight will relocate a locked dislocation without accompanying and proper adjustment of the line of traction. When this is provided, 5 pounds of traction will move the articulating facets as efficiently and more accurately than greater weights. This has been described in detail elsewhere.[1]

If a block is absent there is no need for operation, and x-ray films should be postponed until they can be taken without risk to the patient.

The patient should be placed on an ordinary hair mattress in a bed that is made up in the usual

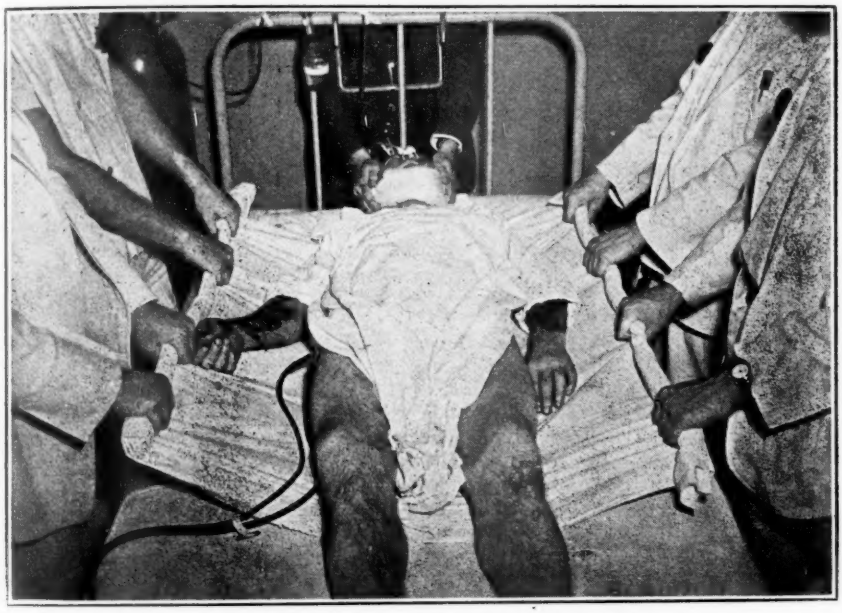

FIGURE 4. *Moving a Patient in Bed with the Aid of a Drawsheet. Note the sheet stretched tightly beneath the patient.*

way. This brings a wide linen drawsheet beneath the buttocks and back (Fig. 4). It is this drawsheet that should be used to turn the patient at the end of every two-hour period. With an ordinary adult it requires at least three and better

The patient's feet should be supported at right angles, with the bedclothes held off them by a cradle, and the knees should be supported on pillows in slight flexion. Particular pains should be taken to see that the paralyzed hands and arms are

FIGURE 5. *Moving a Patient in Bed with the Aid of a Drawsheet.*

The patient is partially turned toward his left side. Note that the patient's head is rotated simultaneously.

five persons to do this. Each of the lateral edges of the drawsheet is grasped by one or two nurses and the sheet is pulled tight underneath the patient. Another nurse holds the patient's head face-up between her hands (Fig. 5). If the patient is to be turned from his back to his left side, the nurse or nurses holding the drawsheet edge to the right of the patient, bring it upward and toward the other side of the bed until it is in contact with the patient's back and side. The drawsheet is kept stretched tightly at all times. A continuation of the movement rolls the patient onto his left side. Simultaneously the nurse holding the patient's head turns the latter in concert with the body (Fig. 6). Once the rotation is accomplished, the drawsheet is used to lift and move the patient back to the middle of the bed (Fig. 7). The traction is not disturbed. To move the patient from his side to his back the sheet is brought to the last position described above, and with this as a starting point the rest of the process is done in reverse. Every patient with any kind of spinal-cord injury can be moved safely as often as need be by this method. It is much safer and more efficient than the use of Bradford frames or any similar apparatus.

not allowed to get into awkward or strained positions or to get beneath the body. They should also be kept off the chest and particularly the abdomen — not because of damage to the extremities but because their dead weight will seriously impede respiration. This is especially dangerous and may make the difference between life and death during the early period after the injury when respiration is either wholly or mostly diaphragmatic. Physiotherapy should be begun and maintained as long as the patient's muscles are not hypertonic. If they are, it is harmful and contraindicated. The latter situation, however, scarcely ever arises in cervical-cord injuries. When it does occur, it is usually a late manifestation of transection of the cord.

Care of the hands is particularly important in all these cases. In many it is also particularly difficult. This is especially true in alcoholic patients or others who have had an inadequate diet, in manual laborers and in patients over fifty years of age. In many of these patients the small muscles of the hand atrophy early, a general swelling of the subcutaneous tissues of the palm and back of the hand and the areas around the finger joints takes place, and stiffness and immobility of the

joints, and in some cases intra-articular adhesions, develop. These changes reach their maximum height in about three weeks after the injury. They make their appearance and persist in spite of adequate and early physiotherapy, although without it they appear earlier and are worse. The hands have a characteristic flat appearance and are best described by the appellation "flippers." As a result, although innervation of these small muscles is complete at the usual time and in accordance with the amount and degree of recovery from the cord injury, function fails to return — perhaps permanently — because of the soft-tissue swelling and the immobility of the joints.

Because of my belief that this might be an "arthritis problem," Dr. Francis C. Hall was asked to see certain patients suffering from this condition. It was his opinion that the lesion was neither arthritic nor periarthritic in the usual sense of the word. He suggested that it had its origin in a combination of subclinical avitaminosis and impairment of the circulation in the upper extremities. For the avitaminosis he suggested the following: eight yeast tablets or one teaspoonful of Vegex three times a day, 3 cc. of crude liver extract twice a week, one tablet of Lederle's vitamin B complex every hour for twelve doses daily for two to three weeks and the avoidance of catharsis when possible. For the circulatory deficiency he suggested diathermy three times a week through the back over the origins of the brachial arteries.

Three patients with the above signs, two of whom were alcoholic, over fifty-five, manual laborers and suffering from hematomyelia, and who had had advanced and uncorrectable disability of this type for seven and three months respectively,

FIGURE 6. *Moving a Patient in Bed with the Aid of a Drawsheet. The patient has been turned so that he lies on his left side.*

were put on the above regime. The third patient was nonalcoholic, over sixty, a foreman, and was suffering from a cervicodorsal transection; he had had the same disability for three months but to a lesser degree than the others. Within a month the hands of all these patients had regained their normal appearance except for the atrophy of the small muscles; the swelling was completely gone, the local circulation was noticeably improved, and the mobility of the joints and consequent usefulness of the fingers had increased by 30 to 50 per cent. During this time every application of the diathermy had caused pain in the shoulders and down the arms. Since then the improvement has continued, but more slowly, and this pain has disappeared. One other patient — a woman of forty-eight, alcoholic and a manual laborer with a hematomyelia — was treated in this way from the start. Four months after injury she had had no more than the expected trouble in regaining the usefulness of her hands and had never shown any of the signs listed above, although she was believed from the start to be a patient who under other circumstances would have certainly developed disability from this cause. Although many more observations must be made and many more of such patients seen

and studied before even tentative conclusions can be reached, the experience seems sufficiently striking so that the profession at large should have the benefit of it.

At the end of six weeks in a case in which there has been bone injury and there is adequate evidence of repair by x-ray, or after eight weeks in those in which there has been minimal or no evidence of bone injury, the patients can be allowed out of bed after being fitted with a modified Zimmer brace (Fig. 8).[4] Elderly patients can be allowed out of bed as early as two weeks after the injury if their general condition warrants it and they wear one of these braces. This practice is justifiable only under special circumstances, however, and should not be applied as a general rule. By the end of six or eight weeks and usually earlier, those patients who have had hematomyelia regain normal control of the bladder and bowels. Constant x-ray check should be made of their bone injury until such time as the physician is certain that it is completely healed. The splint should be worn constantly during this period. This is doubly important if the injury has been ligamentous. Check by x-ray for redislocation in such cases should be frequent and should be continued for four months after the splint has been removed. Evidence that redislocation is developing necessitates the return of the patient to traction in bed and more prolonged splinting.

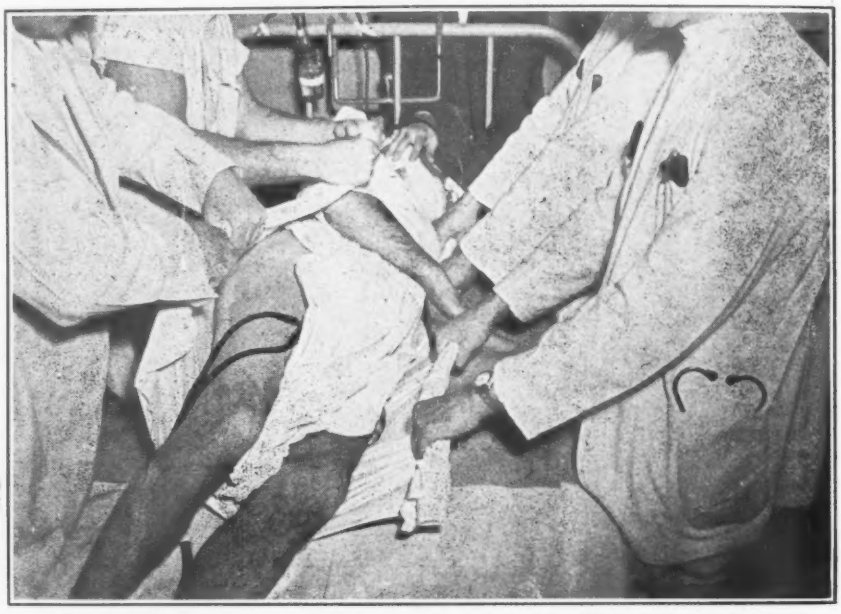

FIGURE 7. *Moving a Patient in Bed with the Aid of a Drawsheet. The patient is moved back to the center of the bed.*

juries. Practically all patients with transected cervical cords die within the first week after their injury. This is particularly true with complete transections. I have never seen a patient with a complete cervical transection develop a mass reflex. As a result, tidal drainage has been adequate and efficient in the care of their bladders. The 2 patients with partial transections who have survived for a longer period have flexion and adductor spasm but none of the excessive motor response to minimal sensory stimuli that characterize the next stage in complete transections and that are the chief characteristic of the mass reflex. The greatest problem in these cervical transections has been caring for the distention that under other circumstances would do little or no harm. In these patients respiration is entirely diaphragmatic. The increased intra-abdominal pressure inherent in distention splints the diaphragm, with a resultant decrease in respiratory efficiency, the development of pulmonary disease and death. Avoidance of fluids, including water, by mouth for the first three to five days and the use of rectal tubes, milk and molasses enemas, physostigmine and similar drugs and procedures, repeated frequently or used constantly, are the best means of

I have never seen any harm done by too prolonged splinting in cases of cervical-cord injury, but have seen unnecessary invalidism develop because of too short a period of both traction and splinting.

The problems inherent in transection of the cord arise only infrequently in cervical-cord in-

combating this condition. Abdominal operation is contraindicated and will kill the patient if performed.

Operative Treatment

In patients who have a positive Queckenstedt test the question arises whether this block should be relieved by a decompressive laminectomy, and if so how soon after the injury. This procedure is not to be undertaken lightly. A comparative study of two similar groups of cervical-cord injuries demonstrated that in 30 patients 43 per cent of whom were operated on, the mortality was 63 per cent, whereas in 46 patients 15 per cent of whom were operated on it was only 33 per cent. In other words, when the number of decompressive laminectomies was reduced by 28 per cent the mortality was reduced by 30 per cent. All these patients are included in the present series. A study of the lesion, as noted above, points in the same direction. Those conditions (contusion, edema and compression of the cord) that at first sight are most in need of early decompression are the ones that end, whether operated on or not, with transection or at least a more serious pathologic condition than was originally believed to have been present. Operation therefore can only do harm unless it is postponed until the changes produced in the cord by the injury have become stabilized. This does not mean that other methods of treatment should be abandoned during this period, nor does it mean that decompression may not prove helpful after the period has passed. It does mean that operative decompression during the first five to seven days after the injury is not justified, regardless of the symptoms and signs of block. During this period the surgeon must depend on properly adjusted traction and improvement in the oxygenation of the tissues to reduce to a minimum any permanent damage to the cord. If at the end of this time a complete block is still present or a partial one is increasing and the patient's general condition warrants it, decompression should be done. It should be extensive enough to free the block completely and the operation should be limited to this procedure only.

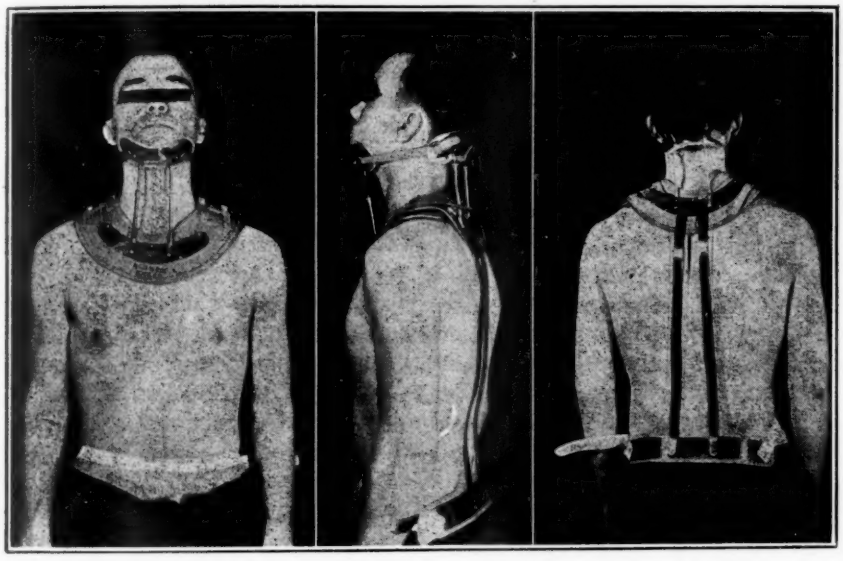

FIGURE 8. *The Modified Zimmer Brace.*

I am strongly opposed in these cases of cervical-cord injury to any attempt to manipulate the damaged bones as part of the operative procedure. The cord has been sufficiently endangered already without adding the chance of injury from a slipped instrument, a loose fragment that had not been seen or a redislocation during closure because of an unrecognized partial reduction. Any necessary and possible change in the bony alignment can be better accomplished by properly applied traction. I am opposed also to the use of silver wire or other material for the purpose of tying one spinous process to another in the belief that this will control the movements of the damaged vertebra or vertebras. Any control comes from proper reduction and sufficient splinting, and in spite of and not because of a loop of wire. Furthermore, the presence of wire is not necessarily innocuous. I have removed one that was surrounded by dense fibrous tissue continuous with a scar in the canal that deformed the dura, the cord and the roots over three segments on one side. The resultant symptoms were disabling.

If possible it is best to use Avertin and local

anesthesia. If the condition of the patient is such as to preclude the use of local anesthesia, Avertin followed by Pentothal Sodium intravenously is the next choice and is preferable to an inhalation anesthetic. Its administration should not be attempted, however, unless a cannula is inserted and tied into an ankle vein and kept open by dripping 5 per cent glucose in salt solution into it. The Pentothal Sodium should be administered by puncturing the rubber tubing of this system close to the cannula with a needle attached to the syringe containing the drug. If the drug is given slowly and repeated often enough, there will be no trouble with the respiration.

Before a final decision can be made as to operability, information relative to the bone condition must be obtained. This should be done as soon as it is known that a block is present. Thus, in blocked cases x-ray examination is essential some time during the first seventy-two hours or as soon thereafter as it is possible and safe to make it. This should include a stereoscopic anteroposterior view and right and left stereoscopic oblique views. A single lateral view can be taken, but one should not expect a great deal from it, especially in the lower cervical injuries. It should never be forgotten that a negative x-ray finding in cervical-cord injuries is more important than a positive one in that it suggests ligamentous rather than bone damage. Bullet wounds and stab wounds are compound injuries and as a general rule should be débrided when it is safe to do so. Whether or not the wound is débrided, however, the sulfonamides should be administered by mouth or intravenously and applied locally to the wound. Beyond this, it is impossible to be more specific. Each case must be settled on its merits, always bearing in mind that the preservation of the patient's life is a necessary prerequisite to his recovery from any paralysis he may have. While the surgeon is reaching a decision as to whether and when to operate, or whenever the block is relieved, the care of the cord injury and the patient is the same as that outlined in the previous section.

Transportation

The problem of how and when to move patients with a cervical spinal-cord injury may arise at any time, and experience in relation to it is particularly apposite in view of the widespread public education in first aid. One fundamental factor governs. This is that the patient's head must under no circumstances be flexed on the chest. Pure extension within reason is harmless, and fixation in mild extension is ideal. Flexion is inexcusable, and denotes complete ignorance of the problem at hand. Patients who walked into the hospital have been changed into bedridden invalids with paralysis of all four extremities after their necks have been flexed in the course of a clinical examination. In other cases flexing of the neck in the course of moving patients from the scene of the accident to a litter has resulted in instant death. The danger attendant on any flexion or any possibility of flexion of the head on the chest in actual or potential cervical-cord injuries cannot be emphasized too strongly in view of the directions for moving such patients given in the *American Red Cross First Aid Text-Book*[5] and the sixth volume of *Military Surgical Manuals*, published under the auspices of the National Research Council.[6] The former is the book used for the instruction of first-aid classes. Specific directions are given there, including a photograph, that a victim with an actual or suspected broken neck must be kept with his face up if lying on his back or turned to a face-up position if lying on his face previous to transportation. This is despite the fact that the directions for caring for a broken back call for transportation in the face-down position unless a combination of broken neck and back is suspected, in which case the victim must be carried with his face up. The latter, a recent publication, is intended for the use of the medical departments of the Army and Navy.

Transportation face up is dangerous for several obvious reasons. Many of these patients vomit. They are paralyzed and cannot move or be moved. When they vomit in this position, the vomitus necessarily gravitates to the back of the throat and is almost certain to be inhaled. This causes atelectasis or pneumonia at best and death by strangulation at worst. Attendants are of no help in preventing such a complication because with the patient in this position they cannot move his head without flexing his neck. This is recognized even by the Red Cross as being extremely dangerous. With the patient in the face-down position this situation can be avoided.

Another source of danger is the self-appointed assistant. He is ubiquitous, and his judgment is as bad as his intentions are good. A request by the patient for a drink of water or a more comfortable pillow, or that his head be moved, or similar suggestions by a bystander, will, if gratified, lead to flexion of the head on the chest when the patient is lying face up. In the face-down position, on the other hand, movement of the head is limited to turning or extension. Either one of these is relatively safe and is therapeutic in comparison to flexion. Many patients with cervical-cord injuries are uncooperative and confused, and persist in trying to get off the stretcher. If they are face up they can and may kill themselves in the course of such attempts, whereas

if they are face down their chances of doing themselves harm are much reduced.

Patients with broken necks who have to be moved before they can be laid face down must have some sort of temporary splint applied to the cervical spine. Such persons are those who are held erect or in a sitting position after the accident or who have to be brought out of a confined space. Among the latter are men injured in tanks or in single-seated fighting planes. Attempts to extract these patients without such splinting almost inevitably leads to flexion of the neck and an increase of cord damage as the least serious result, and instant death as the most serious one. Any splint applied to prevent this development must be easily available, and of such a type that it can be wrapped around the neck before the patient is moved. It needs only enough body to give it stiffness for ten or fifteen minutes. A newspaper fulfills these requirements as well as anything else. It should be folded lengthwise in such a way that its width equals the distance between the angle of the lower jaw and the clavicle. It is wrapped around the patient's neck without moving him and held in place with a piece of string. This is a temporary arrangement, and should not be expected to do more than steady the head on the shoulders during the moving. Neither this nor any other kind of splint need be applied to such patients' necks when they are lying face down, and must not be put on when they are lying face up. For the rest, I am in agreement with the recommendations given in the Red Cross manual. They insist that such patients should always be rolled and never lifted, that they be carried on a solid litter or its equivalent that is longer and wider than the patient, and that the neck and head be supported on both sides by the equivalent of sandbags so that the head cannot roll. When the patient is carried face down, one of his arms should be placed in such a position that the forehead rests on the extensor surface of the forearm. To accomplish this the elbow must be bent at right angles and adjusted in such a position that the forearm lies at right angles to the long axis of the body. Unless and until these requirements can be fulfilled, it is much better and infinitely safer to leave the patient where he is, even if this necessitates standing guard over him, redirecting traffic and the like. Certainly these are no cases for amateur handling.

Complications

One of the commonest and usually forgotten or unrecognized complications of cervical-spine and cervical-cord injuries is a craniocerebral injury. Nineteen patients in this series, or 19 per cent, suffered from craniocerebral injuries, and 7 died. The lesion and treatment are no different from those of the craniocerebral injuries that are not complicated by injuries of the cervical spine or cord (Table 1).

Other significant complications of cervical-cord injuries are interference with the return of function of the hands and the conditions traceable to impairment of respiration. The former has been discussed sufficiently above. Impairment of respiration is a necessary consequence of practically any cervical-cord injury. Either the injury itself or spinal shock will at least reduce and may do away altogether with the usefulness of the costal muscles. As a result, all respiratory exchange must depend for a variable period of time on the diaphragm. This muscle is not equipped to carry such a load unaided and without a chance for adjustment. Respiratory irregularities and deficiency result and may go on to the point where death follows. Distention, restlessness, anoxemia, other injuries, necessary or unnecessary operative procedures and the like all further impede the action of the diaphragm. Unfortunately, the general bodily changes that may be associated with such conditions are not well understood. Treatment is therefore purely empirical and not of great avail. Investigation now going on in my clinic will — I hope — eventually settle some of these questions, but it has not proceeded far enough to justify any final conclusions. At the moment we believe that in practically all cervical transections at any level, and in most of the other more severe injuries that involve the cervical cord close to the fifth segment, the diaphragm must provide the motive power for respiratory exchange for a variable period of time — from a few hours up to the rest of the patient's life. During the first week after the injury or until the first effects of spinal shock have worn off, the diaphragm attempts to adjust itself to this unaccustomed load. If this proves impossible, cyanosis, restlessness, an increase in respiratory rate with a decrease in amplitude and signs of fluid or consolidation in the lungs develop. In the later stages confusion and disorientation further complicate the picture, but whether these are caused by anoxemia or something else is not clear. This syndrome is probably traceable to tiring and consequent inefficiency of the diaphragm, with a resultant decrease in respiratory exchange. It usually but not always results in death. In our experience a respirator and other mechanical substitutes for costal breathing are not only of no help but are actually a hindrance because of the impossibility of adjusting the respirator rate to that of the patient. We have not, however, tried paralyzing the patient's respiration entirely and depending on

the respirator as a substitute. In our hands the application and constant maintenance of a tight chest binder, oxygen given through a nasal tube, control of the bowel and bladder, avoidance of all actions that lead to increased exertion on the part of the patient, movement of the patient every two hours from back to side and the use of the barbiturates by mouth or paraldehyde by rectum have proved the most helpful means of controlling this situation. Morphine, any anesthetic or operation, failure to treat the bladder with tidal drainage or to prevent abdominal distention and failure to change the patient's position every two hours increase the chances of a fatal outcome.

Pressure sores and bedsores are relatively infrequent in cervical-cord injuries, and with proper and prompt care, bedsores at least should never occur and pressure sores should be reduced to a minimum. The points of differentiation, treatment and usual sites of occurrence of these complications have been described in an earlier paper.[7] I shall only repeat the conclusions drawn then and confirmed by further experience. Pressure sores always antedate bedsores. The former develop because of prolonged weight bearing on bony prominences and because of maceration of the horny layers of the skin. The latter develop because of interference with the skin-vascular reflexes. The best treatment of bedsores is the prevention of pressure sores. This is accomplished by keeping the patient constantly dry, turning him every two hours as long as he is bedridden, maintaining the serum protein at 6.0 gm. per 100 cc. or above and the blood chlorides at a normal level, refraining from surgery and dressing the sore only once a day and only with a dry sterile dressing.

End Results

Forty-six of the patients in this series died in the hospital, a mortality of 46 per cent. The causes of death are interesting and significant (Table 5). The commonest cause was respiratory failure. Autopsies have demonstrated that this is caused neither by edema or other intramedullary pathology nor by any primary pulmonary condition. Medullary or brain-stem damage occurs only as the direct result of local injury, and pulmonary disease only as secondary to inadequate respiratory exchange. Further details are not yet apparent. Seventy-four per cent of the deaths were caused by respiratory failure of this kind. Peripheral circulatory failure was the next commonest cause of death. These two together with surgical shock and multiple injuries—that is, conditions traceable to the immediate deleterious effect of the injury—accounted for 87 per cent of the deaths. In this connection it should be noted that 33 per cent of the deaths occurred in the first twenty-four hours after the injury, and 78 per cent in the first week. This emphasizes the importance of the immediate effects of the accident as a cause of death. Comparative figures in thoracic and lumbosacral-cord and conus cases were no deaths from respiratory failure and 22 per cent of deaths from the immediate effect of the accident, only 4 per cent occurring in the first twenty-four hours after the accident and only 9 per cent during the first week.

TABLE 5. *Causes of Death.*

CAUSE OF DEATH	NO. OF DEATHS	INCIDENCE
		%
Respiratory failure	34	74
Circulatory failure	3	7
Surgical shock	2	4
Multiple injuries	1	2
Intercurrent disease:		
Pulmonary embolus	1	2
Phlebitis	1	2
Sepsis:		
Genitourinary tract	3	7
Bedsores	1	2
Total	46	

Sepsis as a cause of death in cervical-cord injuries should not occur if the patient is properly cared for. The 9 per cent of such deaths in this series should have been avoided. Two of the 3 patients who died as the result of sepsis of the genitourinary tract had pyelonephritis. Both antedated the use of tidal drainage. One patient had a cystostomy, and the other had a periurethral abscess following perforation of the urethra with a styleted catheter. This is directly traceable to the failure of the surgeon to recognize that in this and similar cases the urethra is insensitive. One patient died because of septicemia that was associated with extensive pelvic osteomyelitis. This in its turn was caused by a sacral bedsore. This case also antedated tidal drainage. The comparative figure for sepsis in cases with cord injuries below the cervical levels is 43 per cent.

Fifty-five patients have survived (Table 6). Three are still in the hospital and 8 have been lost track of. Six were failures, 1 having died of arteriosclerosis one and a half years after discharge from the hospital to another public institution because of psychosis. All the failures were apparent within three years of their discharge. Twenty patients had a satisfactory result. Five of these have been followed for less than six months, and 1 died of cancer of the rectum between seven and eight years after his discharge. The rest are leading essentially normal, active lives but for various reasons are not earning their living. Eighteen patients are, or were, well; that is, they not only live a satisfactory

normal life but are also self-supporting. Two have been followed for less than six months. Two died, one of intercurrent heart disease and the other of an intercurrent disease the nature of which was unknown but which was reported as being in no way connected with the cord injury. The first patient lived for four to five years after discharge and the second for three to four years.

TABLE 6. *End Results.*

FOLLOW-UP PERIOD	FAILURE	SATISFACTORY	WELL	STILL IN HOSPITAL	LOST
Less than 6 months		5	2	2	7
1 year	5*	3	3	1	
2 years	1	5			1
3 years		4	4†		
4 years		1	2‡		
5 years		1			
6 years			1		
7 years		1§	3		
12 years			2		
15 years			1		
Totals	6	20	18	3	8

*One patient died of arteriosclerosis.
†One patient died of unrelated disease.
‡One patient died of heart disease.
§One patient died of cancer of rectum.

SUMMARY

One hundred and one cases of injury to the cervical spinal cord are reported.

Attention is called to the importance of spinal shock because of its effects on the diagnosis and prognosis; the importance of respiratory failure and sepsis because of their effects on the death rate; the relative unimportance of the bone injury; the significance of ligamentous injuries; the change for the worse that may develop in the severity of the cord injury as originally seen at operation or as deduced from the immediate symptoms; and the diagnostic and prognostic significance of the immediate onset of paralysis following the injury.

The diagnostic possibilities in respect to cord injuries are listed.

A method of treatment as applied to cervical-cord injuries in general and to the cases with and without spinal subarachnoid block in particular is outlined.

The importance of complications arising out of associated craniocerebral injuries, a combination of subclinical avitaminosis and circulatory deficiency with resultant interference with the return of function to the patient's hands, and the impairment of respiration are emphasized.

Attention is drawn to the need for proper care of abdominal distention and the urinary bladder.

The dangers attendant on transportation of patients with cervical-cord injuries in the face-up position are pointed out. An earnest plea is made that such transportation be carried out in the face-down position and that immediate emergency splinting be provided before attempting to move any erect patient who has a broken neck.

The causes of death in the fatalities and the end results in the surviving patients are given.

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PURPURA FULMINANS (THE WATERHOUSE–FRIDERICHSEN SYNDROME)

Report of a Case with Recovery

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THE Waterhouse–Friderichsen syndrome — adrenal hemorrhage associated with purpura — is also known as acute hemorrhagic adrenalitis, suprarenal apoplexy and purpura fulminans. Although the clinical picture was first described in 1901, until recently it has been regarded a very rare condition, and one that is rapidly and invariably fatal.[1] The diagnosis is still not often made clinically.[2, 3] Until February, 1943, there were but 103 cases reported in the literature, and among these there were only 3 recoveries.[3]

With few exceptions the condition is observed in children between the ages of two months and two years. Seventy per cent of all cases fall within this age range, and 90 per cent of all patients are under nine years of age.[3, 4]

The similarity of the course of this disease to that of meningitis and the frequent finding of the meningococcus as the etiologic agent have given rise to a feeling that this is the only cause of the syndrome.[3] Actually, although the meningococcus is the most commonly found agent, there are many possible etiologic factors. In newborn infants, trauma and asphyxia of long and difficult labor, hereditary syphilis and maternal eclampsia have been thought to be implicated.[1] In older children and adults, adrenal hemorrhage has been found to occur in the course of many acute infectious diseases, especially scarlet fever, diphtheria, pneumonia and meningococcal meningitis.[3]

The exact cause of adrenal hemorrhage is not known. Anatomically, the vessels of the adrenal glands are characterized by extremely thin sinusoidal walls in close contact with the parenchyma. It is believed that a toxic effect is exerted on the vessel walls, increasing their permeability and resulting in extravasation of blood.[3] Venous thrombosis is also seen, but this is believed to be a secondary effect. The hemorrhage is located chiefly in the medullary portion of the gland and extends into the cortex, mostly by contiguity. Adrenal hemorrhage is bilateral in 95 per cent of cases, and when unilateral the right suprarenal is practically always involved. Grossly the gland may be enlarged in cases of severe hemorrhage, but its contour is usually not changed.[1] Variations in severity of hemorrhage from petechiae to gross extravasation are directly correlated with the severity of the clinical picture.[5]

Previous to the last few years the clinical picture has not been well recognized,[4] and the diagnosis has rarely been made before autopsy.[1, 2] Features described by observers reveal that the general picture varies considerably, but after the onset, with actual adrenal damage, the pattern becomes clearer and more typical and should be recognized, or at least suspected, oftener than it has been.

The initial symptoms are the nonspecific ones encountered at the onset of any infectious disease. Malaise, fatigue, headache, digestive disturbances and a slight rise in temperature are common. Frequently there is an accompanying upper-respiratory infection. A more suggestive but less frequently encountered train of symptoms is that which occurs when a previously well child awakens during the night with a cry and is seized with nausea, vomiting and pain, followed by mild diarrhea. These mild premonitory symptoms are usually not regarded seriously, but soon the fulminating course characteristic of adrenal hemorrhage appears, and this frequently proceeds rapidly to a fatal termination.

Cyanosis first appears. This may be constant or intermittent, and either generalized or limited to the lips and nails. Usually the appearance of this sign is accompanied by a rapid rise in temperature, but the temperature may not rise until later and may even be subnormal. Within a short time petechial mottling of any or all parts of the body appears. The petechiae are reddish and tend to increase and coalesce, especially about the hands and smaller joints of the body. Lesions of the mucous membrane and conjunctivas are often encountered. Frequently a post-mortem type of lividity is present in the dependent portions of the body. As the condition progresses the patient lapses into a state of profound shock, with a fast, irregular pulse, rapid respirations and a fall in blood pressure. A characteristic feature of shock of adrenal origin is that the extremities become cold whereas the body remains warm.[1] Commonly observed signs of the final phase consist of Cheyne–Stokes respiration, coma, delirium or convulsions, a sudden rise or fall in temparature and death, which may come on in twelve to

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twenty-four hours after the first mild symptoms have made their appearance.

Physical examination in the early stages is discouragingly negative. There may be a few moist rales at the base of the lungs. The abdomen is negative. Neurologic examination and spinal-fluid tests as a rule give little or no positive findings. Blood culture is positive in those cases of infectious origin. Lumbar puncture may be positive in meningococcal cases shortly before death; usually death intervenes before typical findings of meningitis appear. A leukocytosis, the average white cell count being about 14,000, is found. Bleeding, clotting and prothrombin times are normal. The platelet count is slightly below normal.

In view of the small number of reported recoveries, it is believed that the following case in which the outcome was favorable will be of interest.

Case Report

A 19-year-old, recently married girl worked in a defense plant until the day before entry to the Newton Hospital. Three days previous to entry she experienced the onset of a mild, upper respiratory infection. No systemic effects were noticed, and coryza and nasal obstruction were the only complaints. She felt perfectly well, and even these minor complaints were alleviated the day after they were first noticed. She continued her work without the appearance of further symptoms until the day before entry, when she noticed the onset of a mild headache in the midmorning. She took some Emagrin tablets without significant relief. As she continued her work she became conscious of general malaise, but had no specific complaints other than the continued headache, which had localized in the occipital region and had become somewhat more severe. In the midafternoon she reported to the factory medical department, where her temperature was found to be 101°F. Because of this she was sent home to bed. Her vague, general discomfort continued and early in the evening she began to vomit. During the night she had eight bouts of vomiting, which were followed by loose, watery dejections. She also experienced the onset of aching pains located chiefly in the buttocks, ankles, shoulders and tibias. The occipital headache continued to be moderately severe, but there was no cervical pain or rigidity. At midnight, reddish spots, later becoming bluish, began to appear on the abdomen. The patient was seen by a physician, who noted no further physical findings. He saw her again early on the following morning, when the petechial spots had become generalized and were beginning to enlarge and coalesce. The bed was somewhat bloodstained from rectal bleeding. Her temperature was normal, and aside from the petechiae the physical findings were negative.

When first seen in the Newton Hospital on February 19, 1943, the patient presented the picture of a well-developed and well-nourished girl lying in bed in moderately severe pain that was generalized but chiefly localized in the bones. She was conscious, alert and rational. The skin over the lower arms and legs was tan. This was ascribed by the patient to "last summer's tan." The skin was chiefly characterized by a generalized, blotchy petechial eruption. The individual lesions were morbilliform and had no particular pattern of distribution other than that they tended to be concentrated in the extremities. They were of varying sizes, did not blanch on pressure, and were reddish blue. A few petechiae were present on the buccal and pharyngeal mucous membranes and on the scleras and conjunctivas. The heart, lungs and abdomen were normal. There was marked tenderness over the long bones and about the joints. Motion of the extremities caused pain. The ankle jerks were absent. There was no response to the Babinski test. The Kernig and Brudzinski tests were negative. A Rumpel–Leede test was negative.

In spite of the fact that the blood pressure was 60/40, the pulse 150, and the rectal temperature 98°F., the patient did not present a clinical picture of shock. She ate a hearty breakfast soon after entry.

The initial blood examination showed a hemoglobin of 98 per cent, a red-cell count of 5,300,000 and a white cell count of 41,100, with 72 per cent nonsegmented neutrophils, 22 per cent segmented neutrophils, 3 per cent lymphocytes and 3 per cent monocytes. The platelets were grossly observed to be somewhat decreased. The initial urine showed a very large trace of albumin, and the sediment 5 to 7 white cells and 5 to 7 granular casts per high-power field. The bleeding time was 5 minutes, the clotting time 3½ minutes, and the prothrombin time 19.5 seconds.

The patient was placed on shock blocks and given 500 cc. of plasma, without any significant effect on the blood pressure. When the blood count was reported a diagnosis of Waterhouse–Friderichsen syndrome was made. The patient was then given 5 gm. of sodium sulfadiazine intravenously in 100 cc. of saline solution. Two grams of sulfadiazine every 4 hours was begun by mouth 4 hours later. Twenty cubic centimeters of adrenocortical extract was given intravenously following the sulfadiazine. This was followed by 2000 cc. of normal saline solution intravenously.

Four hours after admission the patient's appearance had markedly changed. She was no longer alert but was drowsy and semicomatose, although she could be temporarily roused when questioned. The extremities were cold and numb and a mild degree of cyanosis was present. At that time the blood pressure was 65/40, and the pulse 150. One hour after the administration of the cortical extract the blood pressure had risen to 80/60, the pulse was 160 and weak, and the rectal temperature was 101°F.

On the evening of admission facial edema was evident. Because of this the patient was taken off shock blocks and given cortical extract in 10-cc. doses twice during the night to increase the blood pressure. At midnight the blood pressure was 80/46, and the pulse 140, and regular and stronger. A liter of 5 per cent glucose in saline solution was given during the night. The patient was restless, complained of bony pain and was semicomatose.

On the 2nd hospital day the rectal temperature was 99°F., the blood pressure 90/50, and the pulse 130. The patient was restless and delirious, her head was retracted, and there was definite cervical rigidity. The cyanosis and coldness of the extremities, however, had disappeared. A lumbar puncture revealed a cloudy and slightly thickened spinal fluid. The initial pressure was 195 mm. The spinal-fluid cell count was 1000 red cells and 7550 white cells; the glucose was 32.8 mg. per 100 cc. The

sulfadiazine level of the spinal fluid was 11.6 mg. per 100 cc. No organisms could be cultured.

On admission the blood chloride was 514 mg. per 100 cc., the glucose 94 mg., and the nonprotein nitrogen 116 mg. A blood culture showed meningococci.

The urinary output during the first 24 hours was 15 cc. despite a 4000-cc. fluid intake. Catheterization produced only another 90 cc. That night the subcutaneous tissues were turgid and edematous and the output for the day was only 180 cc. Urinalysis that evening showed a heavy trace of albumin and large numbers of red cells. The nonprotein nitrogen was 103 mg. per 100 cc. Because of the possibility of large amounts of adrenocortical extract in conjunction with large amounts of saline causing fluid retention, these were stopped. Sulfadiazine anuria could not be ruled out, although this was not thought to be likely, but sulfonamide therapy was discontinued. Cystoscopy and ureteral catheterizations were negative. Following this the patient was given 105 cc. of polyvalent antimeningococcus serum. She became more edematous during the night and had periods of apnea and cyanosis. For this Coramine and caffein were given intramuscularly.

On the 3rd hospital day the patient had marked and generalized edema. The urinary output had been 180 cc. during the night. She was unable to hear and had bilateral internal strabismus. The urinary output improved that day. The petechial spots, which had become greatly enlarged and coalescent, began to fade at the periphery. The systolic blood pressure had risen to 140, and the pulse was 90.

The course from this time on was one of gradual improvement without further serious complications. Six days following the administration of the antimeningococcus serum, polyarticular hydrathrosis involving the knees and elbows appeared. Culture of the joint fluid was negative. The only other complications were the development of deep pressure sores over some of the petechial eruption under the heels and buttocks.

The patient had some residual joint stiffness, which improved with physiotherapy. At the time of discharge, 6 weeks after admission, she still had some stiffness in the knees, a slight hearing impairment and a mild degree of myopia. The spinal fluid was normal aside from an elevated protein value—75 mg. per 100 cc. There were still two deep pressure sores on the heels that had not filled in. She felt well.

On June 9, the patient reported for a check on her spinal fluid. The cell count, glucose and protein were normal. The hearing and vision had improved. The pressure sores were well filled in and there were no more joint symptoms. The patient had been at work for a week, and looked and felt well.

Summary and Conclusions

A case of purpura fulminans has been reported because of its rarity and especially because of the extremely low incidence of recovery.

It is evident that the syndrome is being oftener recognized clinically. Any patient, especially a young child, who develops spontaneously or in the course of a contagious disease cyanosis, purpuric eruption, profound shock and hyperpyrexia should be regarded as manifesting the syndrome.

Since the course of this disease is often rapid and fulminating, it is imperative that a prompt diagnosis followed by early and active treatment be carried out. In view of the high mortality, heroic measures seem justified.

Since the infection is usually bacterial in origin, the sulfonamides seem to be the mainstay of therapy.

Hormonal replacement with both adrenocortical extract and adrenalin are indicated in the hope of carrying the patient along until a hoped-for resumption of adrenal function takes place.

Plasma, blood transfusions and other supportive therapy may be indicated.

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INSTRUMENTAL REMOVAL OF A TWO-AND-A-HALF-POUND BLADDER CALCULUS, WITH RECOVERY*

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NEW HAVEN, CONNECTICUT

THE following case is reported because it records the largest bladder calculus ever removed with complete recovery. The dry weight of the calculus was 1134 gm. (2½ pounds). It was composed of phosphates and carbonates.

The literature in regard to bladder calculi is copious, and only a few of the references[1-5] are appended. The search through it included the domestic and foreign literature and was conducted through the Yale Medical Library with microfilm material obtained from other sources, notably the Army Medical Library and the New York Academy of Medicine.

The next largest stone reported as removed with survival is that of Smith,[5] which weighed 2 pounds, 6½ ounces. No follow-up was given beyond the hospital stay. The 4-pound stone reported by Randall[4] is the largest bladder calculus removed during life; the patient died thirty-six hours later.

CASE REPORT

The patient, a 34-year-old man, was admitted to the New Haven Hospital on October 28, 1940, with a chief complaint of pus in the urine. He had had urinary difficulty for the last 4 years, characterized by hesitancy, slight dysuria, nocturia (one or two times), variation in the size of the stream, cloudy urine and occasional hematuria. He had recently lost 23 pounds in weight.

The past history was unimportant except that the patient had had "weak kidneys" as a child. He could recall no specific complaints. The family history was negative.

Physical examination on admission showed a man appearing slightly tired and rather old for his age. The temperature, pulse and respirations were normal. The blood pressure was 156/82. All the teeth were markedly carious. There was a stony-hard, rounded abdominal mass in the suprapubic region that extended almost to the umbilicus. On rectal examination the same mass was felt, as well as a small prostate just within the anus.

The urine was yellow and cloudy, with a specific gravity of 1.015, a slight amount of albumin, no sugar and occasional red cells. It contained innumerable white cells, with many cocci in clumps and many small rods in the methylene blue smear. In the only preoperative determination of the reaction of the urine it was reported as being acid to litmus paper; one of us (R. H. J.), however, knew that the urine was alkaline on at least one occasion.

An intravenous phenolsulfonephthalein test was done. No dye appeared in the urine 10 minutes after injection; 45 per cent appeared in 1 hour and 10 minutes and 30 per cent in 2 hours and 10 minutes. The total appearance of dye in 2 hours and 10 minutes was thus 75 per cent.

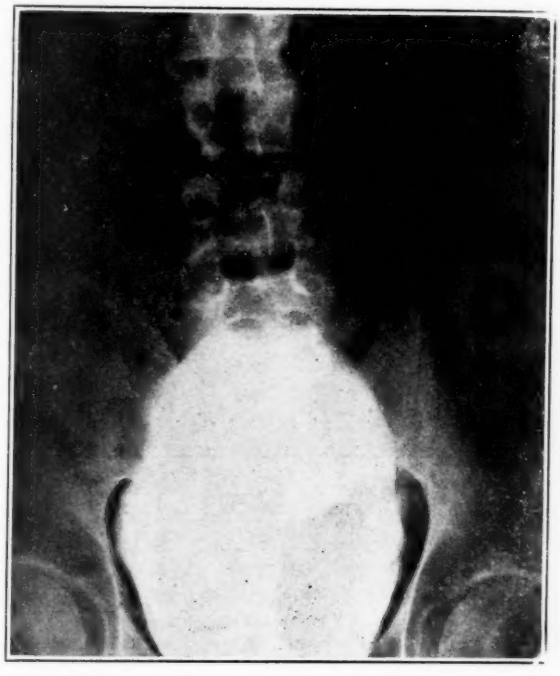

FIGURE 1. *Anteroposterior View of the Calculus.*

The blood was normal. A Kahn test was negative. The nonprotein nitrogen was 31 mg. per 100 cc., the blood calcium 10.5 mg., and the blood phosphorus 3.4 mg.

Intravenous urograms showed bilateral hydronephrosis and hydroureters. There was a huge laminated, calcific shadow in the bladder region (Figs. 1 and 2), but no calcifications in the kidney and ureteral regions.

Eight days after admission, a suprapubic cystolithotomy was done under Avertin, nitrous oxide and oxygen anesthesia. A large, friable, gray calculus was removed with the aid of obstetric forceps, and a suprapubic tube was left in place. At the close of the procedure the blood pressure was 70/60, and the pulse 108. The patient responded well to a 500-cc. blood transfusion, and 5 per cent glucose given intravenously. Two grams of sulfathiazole was given daily for 2 weeks postoperatively, with blood levels between 0.5 and 2.3 mg. per 100 cc. The oral temperature was never above 100.4°F.

The dry calculus was white and flaky, weighed 1134 gm. and was composed of phosphates and carbonates.

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Cystoscopic examination 3 weeks after cystolithotomy showed a slightly constricted vesical neck, which was then resected with a Young's punch. The suprapubic tube was removed and a Foley indwelling catheter was left in place for 5 days. At discharge 10 weeks after operation, the suprapubic sinus was healed and the patient was symptom-free. There was 50-cc. residual urine, which contained many white cells and bacilli.

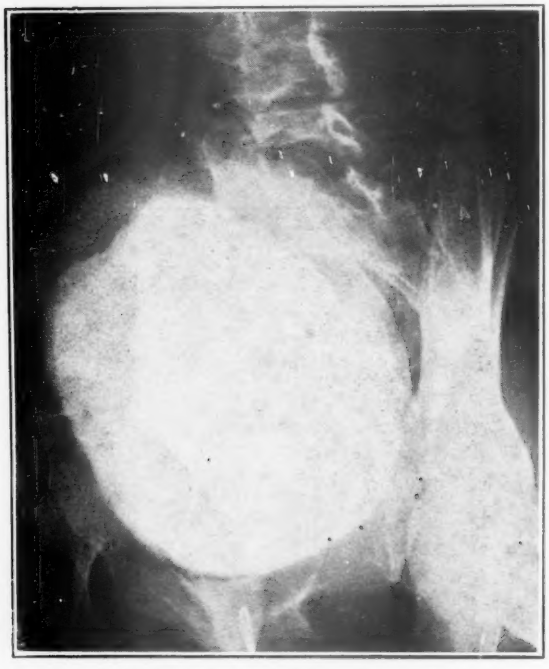

FIGURE 2. *Oblique View of the Calculus.*

After discharge, the patient remained symptom-free and gained 30 pounds in weight. The urine was consistently infected with bacilli until a course of sulfadiazine cleared it in October, 1942, 2 years after removal of the calculus. A complete cystoscopic examination with retrograde pyelograms later in the same month was entirely normal except for trabeculation of the bladder, which had been observed preoperatively. The urine was free from infection, and there was no residual urine. The blood pressure was 140/85. The urine was normal and without infection when the patient was last seen (December, 1942).

In view of the composition of the calculus (analyzed by the methods used by Higgins and Mendenhall[1]), the reaction of the urine was somewhat of a puzzle. Three postoperative checks with nitrazene paper, while the patient was still in the hospital without medication and at various times of the day, showed the reaction to be pH 5.5. When the patient was last cystoscoped the reaction was pH 6.0, as tested with litmus, phenol red, brom-cresol green, brom-cresol purple and nitrazene paper. There is no explanation of this.

SUMMARY

A case report is presented of a thirty-four-year-old man from whom a 2½-pound bladder calculus composed of phosphates and carbonates was successfully removed. The hydronephrosis disappeared, the urinary-tract infection subsequently disappeared, and the patient became symptom-free.

This, to the best of our knowledge, is the largest bladder calculus removed with complete recovery that has been reported.

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Erratum. In the paper "Actinomycosis of the Chest with Spread to the Abdomen" by Drs. Ladd and Bill, which appeared in the November 11 issue of the *Journal*, the abbreviation "mg." should be changed to "gm." in three places: page 749, column 2, line 23, and page 750, column 1, lines 6 and 12.

MEDICAL PROGRESS

CURABLE FORMS OF HEART DISEASE*

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BOSTON

PATIENTS with the conditions discussed in this report comprise only a minority of all those with heart disease. The correct diagnosis, however, is most important, since therapy is highly effective in the majority of cases, whereas in the commoner varieties of heart disease treatment is generally far less satisfactory.

Patent Ductus Arteriosus

In 1939, Gross and Hubbard[1] reported the first successful ligation of patent ductus arteriosus in man; ample confirmation of the usefulness of the procedure is now available.[2–11] In addition, the renewed interest in this condition resulting from Gross's work has stimulated study of the disease.

Etiology. The ductus arteriosus (normally) is patent during intrauterine life; its persistence after birth is therefore not the result of a bizarre congenital malformation.[12] Kennedy[13] recently demonstrated that closure involves two phases previously postulated by Wells.[14] The first is due to a muscular contraction of the wall of the ductus, requiring several minutes; the second phase, obliteration by fibrous tissue, requires approximately one month. Breathing stimulates closure, and Kennedy[15] has shown that the onset of normal breathing in animals is followed by closure in three to ten minutes. Oxygen bubbled into the umbilical vein of fetuses also results in closure of the ductus. He therefore concludes that high concentrations of oxygen in the arterial blood and the beginning of vigorous respiration just after birth presumably combine to promote prompt closure.

Incidence. Patency of the ductus is not rare. In the course of a few years Gross[16] has operated on 56 patients with this condition. Keys and Shapiro[8, 17] have studied 51 patients, and estimate that there are at least 20,000 persons with patent ductus arteriosus in the United States at the present time.

Clinical picture. Patent ductus arteriosus may be recognized clinically by the presence of a so-called "machinery" or constant murmur in the second left interspace, a systolic thrill in this area, dullness in the second or third left intercostal space and the peripheral signs of aortic insufficiency. X-ray study reveals a prominent conus shadow and, in almost all patients, some degree of dilatation of the left auricle, enlargement of the left ventricle, increased pulsations of the left ventricle and pulmonary artery, hilus dance and pulmonary congestion.[4, 18] Electrocardiograms may show left-axis or right-axis deviation or none; when right-axis deviation is present, one must exclude the presence of some other congenital lesion.

Dynamics of the circulation. In man the diastolic blood pressure is usually low and the pulse pressure wide; mild exercise results in a further decrease of the diastolic pressure.[19] No change in mean blood pressure occurs in animals with a long-standing fistula between the aorta and pulmonary artery.[20] The venous pressure in man shows no constant changes.[19] The circulation time is somewhat longer than would be expected in the absence of congestive failure, and is explained on the basis of an increased amount of blood in the lungs.[19] The vital capacity is unchanged. In animals an increase in total circulating blood volume is observed after the establishment of an aortic-pulmonary artery fistula.[19] The cardiac output is increased as a result of a left-ventricular output two to four times higher than the right-ventricular output.[19–21] Since the flow of blood under ordinary conditions is entirely from the aorta into the pulmonary artery, cyanosis is absent. During coughing, laughing, crying, straining at stool and so forth, however, changes in pulmonary intravascular pressure may cause the flow through the ductus to be reversed, resulting in transitory cyanosis.

Results of surgery on dynamics of circulation. At the moment of closure of a patent ductus, the diastolic blood pressure immediately increases,[18–20] probably owing to the diversion of the entire left-ventricular output into the aorta. The pulse rate slows, either because of increased vagal tone or, more likely, as a concomitant of the decrease in cardiac output.[22, 23] There is a decrease and equal-

The articles in the medical-progress series of 1941 have been published in book form (*Medical Progress Annual*. Volume III. 678 pp. Springfield, Illinois: Charles C Thomas, 1942. $5.00).

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ization of the cardiac output of both ventricles,[19] lasting for at least twenty-seven minutes.[20] Blood samples taken from the pulmonary artery show a more marked degree of oxygen unsaturation than was present before closure.[20] The above-mentioned increases in circulation time and blood volume are restored toward normal following closure of the ductus.[19]

Clinical results of surgery. Reports by Burwell, Eppinger and Gross[18, 19] indicate that enlargement of the heart due to dilatation, when present, disappears rapidly following surgery. Hypertrophic enlargement changes little in the immediate postoperative period. The cardiothoracic ratio, however, eventually becomes normal. Patients with definite congestive failure preoperatively show no symptoms of failure postoperatively despite a greatly increased physical activity.

The mortality of operation is low. Gross[16] reported 2 surgical deaths in 56 operations. Complete division of the ductus, in addition to ligation, has been successfully accomplished in his last 14 cases. The advantage of division is the assurance of complete and permanent closure of the ductus.

Indications for surgical closure of patent ductus arteriosus. The majority of patients with patent ductus arteriosus die of cardiovascular complications; 25 per cent die with congestive failure, 25 to 33 per cent die with bacterial endarteritis, and 12 per cent die suddenly.[24]

Burwell[25] has recently stated:

> I think that any expression of opinion at this stage of our knowledge is bound to be tentative since we have been able to observe patients for only a few years after closure of the ductus arteriosus by operation. Two statements may be made:
>
> First, the presence of important limitation or actual heart failure, the demonstration that the patent ductus is interfering with normal growth, or the presence of subacute bacterial endarteritis would seem to me to constitute firm indication for the operation.*
>
> Second, because of the probable utility of the operation in preventing progressive overwork of the heart and in preventing bacterial endarteritis in a young person, I should consider the demonstration of uncomplicated patency to be in itself a strong suggestion that operation should be considered.

Subacute Bacterial Endarteritis Superimposed on Patent Ductus Arteriosus

Incidence. The occurrence of superimposed infection in patent ductus arteriosus is frequent. Reports[8, 24, 20] indicate that from 20 to 40 per cent of all patients in whom the condition is recognized die with this complication.

*This statement is in agreement with the published reports of many observers.[26–28]

Diagnosis. The diagnosis is based on evidence pointing to the existence of patent ductus arteriosus: generalized evidence of toxemia, such as fever, tachycardia, leukocytosis, weakness and pallor[30]; and the symptoms and signs of bacterial endocarditis — that is, splenomegaly and clubbing of the fingers. The course may be that of a protracted atypical pulmonary infection due to repeated small pulmonary emboli[31] or may be characterized by manifestations of peripheral embolism, or both may occur. Peripheral blood cultures may be negative until the lungs are unable to filter out all the organisms, the pulmonary lesions break down and release organisms into the peripheral blood stream, and spread of the infection occurs to the greater circulation.

Therapy. Although it is not certain that surgical ligation or division of the ductus will prevent the subsequent development of bacterial endarteritis, this may well prove to be the case. To date there have been 3 cases of infection developing on a previously ligated ductus. One patient was reoperated on successfully[32]; 2 died following the initial operation.[4, 33] Graybiel et al.[34] reported the first attempt at surgical cure of subacute bacterial endarteritis complicating patent ductus arteriosus. At approximately the same time Keele[35] and Touroff[36] reported similar cases. Touroff has reported operations on 11 such patients thus far.[27, 28, 37, 38] Of the 9 patients surviving the operation, 6 recovered from the infection. Burwell and Gross[39] have recorded the findings in 7 patients with patent ductus arteriosus and superimposed bacterial endarteritis. All were treated with chemotherapy — sulfapyridine, sulfathiazole or sulfadiazine — and with surgical closure of the ductus. One patient was cured on a medical regime before surgery was undertaken. The remaining 6 patients still had positive blood cultures when surgical closure of the ductus was performed. Two of these received no benefit from the combined medical and surgical therapy and subsequently died of their infection. The 4 remaining patients have apparently been completely cured, as evidenced by cessation of fever, repeatedly negative blood cultures, gain in weight and a resumption of normal activities. An additional case has been reported in which chemotherapy alone resulted in cure.[40] It is of interest that there have been several reports of cure of subacute *Streptococcus viridans* septicemia by excision of an arteriovenous fistula.[41, 42]

It is evident that a higher percentage — approximately 67 per cent — of recoveries can be expected following chemotherapy and surgery together[43–46] than has been reported from chemotherapy used alone or with heparin, or from any

other type of medical regime.[47, 48] It may be concluded that when intensive treatment with the sulfonamides does not result in the disappearance of infection, both clinically and bacteriologically, within a short period of time — that is, approximately two weeks — surgical ligation of the ductus is indicated.

The contraindications to surgery are evidence that the patent ductus arteriosus compensates for another coexisting congenital cardiovascular anomaly, and evidence that the vegetations have already spread to the aortic or mitral valves. The presence of soft new murmurs or a positive peripheral blood culture (a few colonies) cannot, however, be accepted as absolute evidence of spread. It is clear that early recognition and prompt therapy will minimize its likelihood.

Mechanism of action of surgery. It has been shown that shortly after surgery blood cultures become sterile.[27, 41] In some cases this has been true of the first blood culture taken ten minutes after ligation. It is possible that the lung acts normally as an efficient filter, since blood cultures from the pulmonary artery contain many more colonies than do those obtained from the aorta.[49] The efficiency of the lung as a filter may increase when the traumatizing effect of the higher aortic pressure on the lesion in the ductus or pulmonary artery is removed by ligation. It is also possible that the decrease in pulmonary-artery pressure, the slowing of the pulmonary-artery blood flow and the decrease in oxygen content of the pulmonary arterial blood are factors.

The Heart in Nutritional Deficiency States

Although thiamine deficiency is a well-recognized cause of congestive heart failure,[50–53] it is perhaps not so widely recognized that deficiency states may exist in patients with congestive heart failure due to intrinsic cardiac lesions. Anorexia, the dietary limitations, the anoxia and congestion of the gastrointestinal tract leading to impaired absorption and the possibly deficient storage and utilization of the substances comprising the vitamin B complex in the presence of impaired liver function combine to give rise to vitamin B deficiency in many cardiac patients with congestive failure. These patients exhibit a red tongue, cheilosis and absence or diminution of tendon reflexes and vibratory sense in the legs.

Pathologic physiology. Porter and Downs[54] recently pointed out that thiamine deficiency in man results in an increase in cardiac output, both absolute and in relation to the oxygen consumed, an increase in blood volume, a decrease in circulation time, an increase in oxygen consumption and elevation of the venous pressure. The decreased arteriovenous oxygen difference and the decreased circulation time suggest the presence of an abnormally dilated peripheral vascular bed. The vital capacity is low and the blood pressure may be normal. Electrocardiographic changes are noted in almost all patients.[51] These consist of tachycardia, a change in the direction of the T waves and a prolonged QT interval. These abnormalities tend to disappear after adequate administration of thiamine, suggesting the presence of a functional rather than an anatomic disturbance. It has, however, been shown that thiamine deficiency may be followed by focal myocardial necrosis[55, 56] and cardiac hypertrophy.[57]

Therapy. It has been pointed out by Hawes et al.[58] that effective therapy in oriental beriberi with cardiovascular dysfunction requires the use of large doses of vitamin B_1 parenterally. These authors stress the fact that in many cases enormous doses have been given by mouth with hardly any noticeable effect. Patients observed in this hospital support this contention. The following case is illustrative of the therapeutic effect of parenteral administration of thiamine chloride and vitamin B complex.

A. L., a 53-year-old man, entered the Beth Israel Hospital on June 27, 1943, with a history consistent with rheumatic fever at the age of 12. Twenty-seven years before entry he had been refused life insurance because of some cardiac abnormality. Ten years before entry he began to experience increasing fatigability, mild dyspnea on exertion and occasional edema of the ankles. About 1 year before admission these symptoms increased, and during the previous 6 months dyspnea, both exertional and paroxysmal, orthopnea and peripheral edema became severe and progressive. Marked anorexia and a weight loss of 25 pounds were evident during the previous year. Treatment prior to hospitalization consisted of digitalization, intermittent bed rest and injections of Mercupurin. His condition, however, became so severe that he entered the hospital.

The essential findings were as follows: temperature, 99°F.; pulse, 70; respirations, 25; and blood pressure, 160/60. The patient was pale and cachectic, perspiring profusely, and exhibited severe dyspnea and orthopnea. Slight icterus, moderate cyanosis and marked venous engorgement with prominent venous pulsations were evident. There were redness and atrophy of the tongue, as well as cheilosis. The anteroposterior thoracic diameter was increased, and diminished resonance, voice sounds and breath sounds, as well as fine crackling rales, were noted over both lung bases. The heart was tremendously enlarged to both left and right, with a widespread bounding apex impulse in the left axilla. The head bobbed and the chest wall heaved with each heartbeat. Characteristic murmurs of aortic and mitral stenosis and insufficiency were heard. The second aortic tone was almost inaudible. The liver was enlarged a handbreadth below the right costal margin and was firm and moderately tender. Slight clubbing of the fingers, bounding radial pulses and marked pitting edema of the legs, with a small amount of sacral edema, were apparent. The knee

jerk reflexes were sluggish. The Achilles tendon reflexes were not obtained.

Examination of the urine revealed normal findings, except for a slight trace to a trace of albumin during the first days. The blood counts were normal. The blood nonprotein nitrogen was 53 mg. per 100 cc. on admission, rising to 82 mg. during the next 3 days and subsequently returning to normal. The circulation time (calcium gluconate) was 60 seconds. The total blood protein was 6.9 gm. per 100 cc. A 7-foot roentgenogram showed the transverse diameter of the heart to be 21 cm. (twice the expected diameter for the patient's height and weight). The pulmonary arteries were prominent, the left auricle moderately dilated, and the pulmonary vessels dilated. Electrocardiograms showed left-axis deviation, a prolonged PR interval (0.22 seconds), depressed ST_1 and ST_2, inverted T_1 and T_2, and auricular and ventricular premature beats.

On admission oxygen was administered by mask, redigitalization was accomplished, and ammonium chloride and vitamin B complex (6 capsules a day) were given. Dyspnea, marked heat intolerance and apprehension increased. Mercupurin, given intravenously on the 4th day, resulted in a diuresis of 4500 cc. in 24 hours. In spite of this, the course during the next 9 days was downhill. Increasing edema, severe dyspnea at rest, growing intolerance of the summer heat and several psychotic episodes were noted. There was no diuretic response or change in the clinical picture following the subsequent intravenous injection of Mercupurin. On the 13th day all oral vitamin B was omitted, and 100 mg. a day of intramuscular thiamine hydrochloride and 1 cc. of vitamin B complex were begun. Within 36 hours a striking change in the clinical picture took place. The dyspnea became minimal, the very severe heat intolerance disappeared although the weather was warmer, and the patient had no more psychotic episodes. During the course of the next 6 days the pulmonary rales disappeared and the peripheral edema became clinically indetectable following a favorable response to Mercupurin injection. The oxygen tent was omitted 2 days after institution of parenteral vitamin therapy. In addition the patient developed an appetite for food. The parenteral vitamin therapy was omitted after 8 days and vitamin B complex (6 capsules) and 15 mg. of thiamine hydrochloride were given orally. Mild to moderate dyspnea and slight to moderate pitting edema began to appear about 10 days after the omission of the parenteral vitamins. The patient was discharged on the 38th day much improved, but with slight dyspnea at rest and slight pitting edema of the ankles and sacrum.

Equally favorable effects on dyspnea and psychosis have been noted following administration of thiamine hydrochloride and vitamin B complex parenterally in other patients with vitamin B deficiency and severe organic heart disease causing long-standing decompensation with severe dyspnea after failure of the usual treatment with digitalis, diuretics and oxygen to secure improvement.[59, 60] No striking benefit has been noted after the administration of large amounts of vitamin B complex, including thiamine, by mouth for periods from a few weeks to several months or more.[59]

Chronic Constrictive Pericarditis

Chronic constrictive pericarditis is recognized by the presence of a small or only slightly enlarged heart, diminished heart tones, markedly increased venous pressure and an enlarged liver with ascites. Blalock and Burwell[61] and Harrison and White[62] have recently reported follow-up studies on their series of cases of constrictive pericarditis, and conclude that approximately 60 per cent of all cases may be cured by surgical excision of portions of the scar. Although the surgical mortality rate is low,[61, 63] it is of further interest that both groups of investigators stress the point that not all patients need surgery; if the disease is not progressive and the disability is mild, patients do well on salt restriction, diuretics and tapping of serous cavities. Digitalis is of no value except when myocardial weakness or auricular fibrillation is present.[61]

Pathologic physiology. Thickening and contraction of the pericardium or epicardium or both prevent the heart from dilating normally and therefore decrease the amount of blood received during diastole. There may also be some interference with emptying of the heart. It has recently been demonstrated that chronic compression of the heart results in atrophy of disuse, made manifest by smaller muscle fibers.[64] The decreased filling, with increased venous pressure, results in an increase in pulse rate (Bainbridge reflex). The decreased output and inability to increase it normally result in weakness, fatigue, tachycardia, low pulse pressure and a diminished tolerance for exercise. The increase in venous pressure leads to the peripheral signs of venous congestion. The circulation time is prolonged and the arteriovenous oxygen difference[65] is increased. Hitzig[66] has recently offered an explanation of the occurrence of pulsus paradoxus in constrictive pericarditis: If the compression is predominantly right-sided, the phenomenon of pulsus paradoxus appears when there is a marked discrepancy between the pulmonary blood volume during expiration and the pulmonary vascular capacity during inspiration. This results in inadequate left ventricular filling and a decreased output with a weakened beat.

The electrocardiograms in White's[62] series showed normal rhythm in 60 per cent and auricular fibrillation in 37 per cent of patients. Low voltage of the QRS complex was found in 60 per cent, with little change following surgery. Abnormal T waves in two or three of the classic leads were found in all cases; none of the T waves became normal in more than one lead following surgery. The electrocardiogram during pericardiectomy

has been studied by Stewart and Bailey.[67] Premature beats and paroxysms of abnormal rhythm, such as auricular fibrillation, auricular flutter and tachycardia, as well as ventricular tachycardia, were observed. The changes were of short duration and were similar to those previously recorded by Feil and Rossman.[68] Serial electrocardiograms taken for months after operation showed few changes in the form of the T waves or ST segments.[67]

Wounds of the Heart and Vessels

Trauma to the heart. The war has resulted in increased interest in cardiac trauma.[69–73] In such cases the early recognition of cardiac tamponade is most important. A large, quiet heart, an increase in venous pressure, or rapid pulse rate, a drop in blood pressure with a low pulse pressure, cyanosis and orthopnea are evidences of cardiac compression.

The Army[74] has recently formulated the treatment of penetrating heart wounds as follows: Blood must be aspirated from the pericardium by the costoxiphoid route, if possible within two hours of the onset of the tamponade. The advantages of the costoxiphoid route are that the needle avoids passing through both pleural leaves, that one cannot injure the lingula of the upper lobe if it is adherent, and that the pericardial sac can be more satisfactorily cleared of blood.[75] After anesthetizing — with 1 per cent novocain — the skin and underlying tissues, a 15-gauge, short-beveled needle is inserted at the left costoxiphoid junction and directed inward and slightly upward until blood escapes. Either a small syringe or no syringe should be attached to the needle. If the needle touches the heart, a scratching sensation is felt, and the needle must immediately be slightly withdrawn. Paracentesis may be done at the site of injury, if indicated, since even the withdrawal of a small amount of blood may be lifesaving. Several reports are available noting the beneficial effects of conservative treatment by means of aspiration and plasma, blood and saline infusions.[76–78] Recurrence of tamponade is an indication for a second paracentesis. This should not be done less than fifteen minutes after the first in order that closure of the wound by a clot may be favored. If tamponade again recurs, extrapleural cardiorrhaphy is indicated. The mortality rate from this operation in experienced hands varies from 26 to 50 per cent.[71, 76]

It is now accepted that contusion of the heart may be consequent on nonpenetrating trauma to the chest wall. An interesting variant is afforded by a case of intrauterine cardiac injury produced by a nonpenetrating blow to the maternal abdomen, with the death of the fetus.[79] In addition, Scherf and Terranova[80] have recently shown that displacement of the ST segment of the electrocardiogram occurs following blunt trauma to the precordium. The traumatic nonpenetrating wounds of the heart may present the following clinical features: absence of symptoms, with complete recovery; delayed, fatal rupture of the heart; myocardial failure, both transient and persistent; hemopericardium; ruptured valve; coronary thrombosis and acute myocardial infarction; angina pectoris, both transient and persistent; and arrhythmias, both transient and persistent, particularly auricular fibrillation.[81–84] The recognition of the possibility of cardiac damage after blunt trauma to the chest should be emphasized. The absence of bruising or other external evidence on the chest wall should not be construed as excluding cardiac trauma. Adequate treatment in the form of bed rest, oxygen administration, sedation and paracentesis may result in complete clinical recovery.

Traumatic arteriovenous aneurysms. The effects of an arteriovenous fistula on the circulation are similar to those noted above for patent ductus arteriosus. In arteriovenous fistula, however, since the systemic circulation is involved, the ventricular outputs are equal, and an increased blood flow is present in both the systemic and pulmonary circuits. Many case reports of successful repair of such fistulas have been published.[85–87] Occasionally, however, spontaneous healing occurs.[86, 88] It is important to remember in this connection that congestive heart failure may occur months or years after the establishment of an arteriovenous fistula.[87] Surgical removal of the fistula results in disappearance of the signs and symptoms of congestive failure.[86, 87, 89] Reid and McGuire[86] emphasize the importance of prolonged bed rest with elevation of the affected part and postponement of surgery for three to six months. During this time hemorrhage becomes absorbed, the tissues are restored toward normal, the danger of infection is lessened, and collateral circulation develops.

There have been to date two reports of cures of subacute *Streptococcus viridans* septicemia due to an arteriovenous endarteritis by excision of the fistula.[41, 42]

Cardiovascular Syphilis

Cardiovascular syphilis can be prevented in 99 per cent of patients by the prompt and energetic treatment of early syphilis.[90, 91] It is important to recognize that adequate treatment of cardiovascular syphilis is effective only before serious complications arise. The criteria for the diagnosis of uncomplicated syphilitic aortitis are teleroentgenographic and fluoroscopic evidence of aortic dila-

tation; a tympanitic, bell-like, tambour accentuation of the aortic second sound; a history of circulatory embarrassment; increased retromanubrial dullness; progressive cardiac failure; substernal pain; paroxysmal dyspnea; a systolic aortic murmur; and visibly or palpably increased pulsation in the episternal notch. A recent study has shown that 88 per cent of patients with aortic syphilis have a positive serologic reaction.[92] Moore and his associates[93] believe that the presence of three or more of the first seven of these signs or symptoms in a patient with known late syphilis under fifty years of age is strong evidence for the diagnosis of uncomplicated syphilitic aortitis, and that the presence of any two of them renders the diagnosis probable. Recent papers have, however, denied the validity of the above-mentioned criteria.[94, 95] Berk[96] has recently formulated criteria necessary for the diagnosis of syphilitic aortitis with coronary ostial stenosis: an abnormal electrocardiogram with progressive serial changes in the absence of acute myocardial infection, and a normal or borderline electrocardiogram in a syphilitic patient under forty-five years of age that shows marked changes after exercise. Actually, however, a positive diagnosis of cardiovascular syphilis can be made only when one of the following findings is present: a saccular aneurysm of the aorta or innominate artery; aortic regurgitation, appearing for the first time in a middle-aged person with a positive serologic reaction for syphilis; and a diffusely dilated aorta without aortic regurgitation or hypertension, past or present.[97]

Treatment. Treatment in cardiovascular syphilis is essentially an individual problem. Table 1, derived from a recent paper by Wile,[98] summarizes the essential points. The best results are obtained in Group III. There is available ample evidence of the deleterious effects of beginning treatment with, or using large doses of, arsenicals.[99–101]

Thyrotoxic Heart Disease

There is still no agreement whether thyrotoxicosis can induce congestive heart failure in patients with normal hearts. Likoff and Levine[102] recently analyzed the clinical data in a series of 409 thyrotoxic patients; 21 cases of congestive heart failure were found in a group of 331 noncardiac patients. They suggest that vitamin B deficiency may have played a contributory role in the production of heart failure in those cases.

The clinical picture in thyrotoxicosis is well defined and requires no discussion here. It must be remembered, however, that some patients, particularly those above the age of fifty, with thyrotoxic heart disease may be apathetic, listless and weak rather than overactive. The clinical findings of stary eyes, marked intolerance to heat, rapid auricular fibrillation refractive to digitalis and excessive weight loss are suggestive and should lead to studies of the metabolic rate. In occasional cases the syndrome simulates rheumatic heart disease with mitral stenosis because of the presence of rough apical systolic or diastolic murmurs or both, with a snapping first tone. The finding of a normal or rapid circulation time in patients with congestive heart failure should also lead one to suspect the presence of thyrotoxicosis, although it must be remembered that congestive failure due to thiamine deficiency may also be associated with normal or rapid circulation times. In this connection Hurxthal[103] has pointed out that patients over the age of fifty with a basal metabolic rate of +50 per cent and a weight loss of 50 pounds are the worst surgical risks.

Table 1. *Treatment of Cardiovascular Syphilis.*

Classification	Previous Treatment	Signs and Symptoms	Types of Treatment to Be Used
Group I	Energetic	Those of syphilitic aortitis, aneurysm and aortic regurgitation; no congestive failure or angina pectoris.	None
Group II	Energetic	Same as Group I, but with congestive failure or angina pectoris	None
Group III	None or inadequate	Same as Group I	Nonintensive but continuous therapy, with intramuscular bismuth, followed by oral mercury and iodides. Subsequent use of small doses of arsenicals in syphilitic aortitis, but rarely in other forms of cardiovascular syphilis.
Group IV	None or inadequate	Same as Group II	Same as Group III when patient has recovered from congestive failure.

The preoperative care of patients with thyrotoxic heart disease involves two procedures.[104] The first is a reduction of the level of metabolism and thus the work of the heart by rest in bed and the administration of iodine. Thiouracil, which has been successfully used in the treatment of hyperthyroidism,[105, 106] may in time replace iodine. The effect of various sedatives should be determined preoperatively.[107] The second procedure is to increase the efficiency of the heart by the adequate treatment of congestive heart failure. Quinidine is not used preoperatively in patients with auricular fibrillation by the Lahey group,[104] since most patients with fibrillation spontaneously revert postoperatively. Those patients who do

not revert may then be treated with quinidine. On the other hand, the Massachusetts General Hospital group[108] gives quinidine preoperatively, except to those patients with active auricular fibrillation, beginning twenty-four hours before surgery and continuing for twenty-four to thirty-six hours thereafter. They also stress the importance of reassuring the patient regarding the entire operative procedure, the anesthetic, the postoperative discomfort, intravenous injections and the oxygen tent.

Anemia as a Cause of Cardiac Pain and Congestive Failure

An erroneous diagnosis of heart disease is often made in patients with anemia[109] because of dyspnea, easy fatigability, weakness, precordial pain,[110] murmurs that simulate those of mitral stenosis[111] and edema. It is true, however, that severe anemia of any origin may have a harmful effect on a normal or diseased heart because of the persistent marked increase in cardiac work consequent on a decrease in the hemoglobin level below 50 per cent.[112–114] It is sometimes difficult to differentiate dyspnea due solely to an inadequate amount of circulating hemoglobin from that consequent on congestive heart failure in a severely anemic patient. The presence of rales, orthopnea and venous engorgement in the latter condition and their absence in the former[115] are very helpful.

Incidence. A recent study by Riseman[116] has shown that anemia, thyrotoxicosis and polycythemia occurred in his clinic in a total of less than 1 per cent of patients with angina pectoris. There are, however, many patients with anemia whose presenting complaints are those of angina pectoris or congestive failure. Thus, from 1918 to 1937, Stalker[117] collected 100 cases of angina pectoris and pernicious anemia. Since 1937, several reports indicate the not uncommon association of angina pectoris and hypochromic anemia.[115, 118] Congestive failure precipitated by anemia appears to be less common, since only a small number of cases have been recorded.[111, 115, 119]

Pathologic physiology of the circulation. The effects of anemia on the circulation resemble in some ways those produced by thyrotoxicosis or an arteriovenous shunt. The systolic and diastolic blood pressures are lowered,[109] with an increase in pulse pressure probably due to peripheral vasodilatation and a decreased blood viscosity. The circulation time is more rapid,[120, 121] although it may be normal in the presence of congestive failure,[122] and the cardiac output and oxygen utilization are increased.[112, 114] Considerable cardiac enlargement occurs in approximately 50 per cent of patients and is particularly common in the older age group.[109] Although most of the enlargement is due to dilatation, some hypertrophy apparently occurs.[123] The electrocardiograms may show flattened or inverted T waves, depression of the ST segments, particularly in Leads 1 and 2,[109, 124] tachycardia, auricular ventricular nodal rhythm[109] and prolongation of the QT interval.[115] These phenomena are responsible for the tachycardia, dyspnea, increased fatigability, weakness and edema noted in anemic patients. A contributory factor in the genesis of edema may be the decreased blood total protein with a reversal of the albumin-globulin ratio seen in some patients.[115] Although most anemic patients with angina pectoris have underlying organic heart disease, several reports stress the normality of the coronary arteries in such patients.[125, 126] The anemia, with its consequent reduction in oxygen capacity, results in myocardial anoxemia and chest pain.

Response to therapy. It is universally agreed that blood transfusions,[118] iron, liver and bed rest result in cure in almost all cases, whether the symptoms are those of congestive failure or of angina pectoris. The changes in the circulation, size of the heart and electrocardiograms noted above are restored to normal. This of course indicates that the changes induced by anemia are functional rather than organic in nature. Although bed rest may not be necessary for all patients,[127] in some cases of congestive failure its use together with digitalis and diuretics may be required to tide the patient over until the hemoglobin level increases significantly in response to antianemic therapy.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor**

BENJAMIN CASTLEMAN, M.D., *Acting Editor*

EDITH E. PARRIS, *Assistant Editor*

CASE 29501

PRESENTATION OF CASE

A sixty-year-old housewife entered the hospital because of rectal bleeding.

The history was taken through an interpreter and was rather incomplete. The patient had been in good health until about one year prior to admission, when she had an episode of "moderate" rectal bleeding. There was no pain or "any other symptoms." It is not stated how long the bleeding lasted. Since that time she had had two similar episodes. At various other times she had noticed that her stools were blood streaked. Her bowels had always been regular. She had had no diarrhea and rarely took cathartics. Her appetite had been good. There was no vomiting, but occasional regurgitation of food after eating. There had been no weight loss. In the few months preceding entry she had a more or less constant ache in the right side of the abdomen, "just opposite the umbilicus," which was usually accompanied by gaseous distress. Two barium enemas performed at an outside hospital were said to have shown a polypoid filling defect in the lateral wall of the ascending colon just above the ileocecal valve.

She had had a hysterectomy eighteen years before entry, and a right nephrectomy sixteen years prior to admission following an attack of severe right flank pain. No diagnoses were recorded for either operation, and no additional information was available.

Physical examination showed a well-developed, rather obese woman in no distress. The lungs were clear. There was a Grade 2 aortic systolic murmur. The aortic second sound was greater than the pulmonic. The abdomen was not distended. Peristalsis was normal. No masses could be palpated. There was tenderness with some spasm deep in the right side of the abdomen at the level of the umbilicus. The scars of the previous operations were well healed. There were no hernias.

*On leave of absence.

The blood pressure was 140 systolic, 82 diastolic. The temperature, pulse and respirations were normal.

Examination of the blood showed a white-cell count of 10,600, with 56 per cent neutrophils and 39 per cent lymphocytes. The hemoglobin was 13 gm. A blood Hinton test was negative. The urine was negative. The stools were guaiac negative. The blood sugar was 68 mg. per 100 cc., the nonprotein nitrogen 10 mg., and the total protein 7 gm.

An abdominal exploration was performed on the fifth hospital day.

DIFFERENTIAL DIAGNOSIS

DR. RICHARD H. SWEET: I wonder if it was accurate to describe the filling defect as being polypoid. In examining an x-ray film Dr. Holmes has taught us to describe what we see and then go to the diagnosis. I assume there was more than one x-ray observation.

The record does not say whether this patient was admitted under the care of a medical service or under the care of the surgeons. I should not mention that except that a great deal of reliance must be placed in some of these histories on the interpretation of physical findings, and my medical friends are not always certain of their own evaluation of such things as tenderness and spasm in abdominal lesions. They sometimes call the surgeon in. The record does not say, furthermore, whether any x-ray films were taken in this hospital.

DR. BENJAMIN CASTLEMAN: We have the films from the other hospital. Perhaps Dr. Mueller will comment on them now.

DR. H. PETER MUELLER: I examined this patient in an outside hospital and found this lesion, which has the appearance of a polypoid mass (Fig. 1). In addition to this round, filling defect, which appears to be opposite the ileocecal valve, the patient was definitely tender on palpation. I was not quite sure when I saw the films for the first time whether this meant anything, or whether it was just due to an enlarged ileocecal valve. I repeated the x-ray films a few days later and the same findings were observed. I had the impression that the filling defect seen in the spot films was too large for the ileocecal valve and that it was probably due to a polypoid mass.

DR. SWEET: What do you mean by "polypoid"? Do you refer to the configuration of the shadow, or to the fact that the lesion appears to hang from the wall of the bowel?

DR. MUELLER: I had the impression that a mass

with a broad base was projecting from the wall of the bowel into the lumen.

DR. SWEET: Above the level of the cecum in the ascending colon?

DR. MUELLER: Yes.

DR. SWEET: The record states that a mass was felt. The patient was an obese woman; hence it must have been quite a mass if they were able to feel it.

There is not much information in the record, and I can summarize it quickly. The patient had rectal bleeding, but there were periods when she had none. In this hospital the stools showed no blood on chemical examination. That is unlike a carcinoma. I should expect rather constant, at least microscopic or chemical, evidence of blood in the stools in a case of carcinoma of the colon. Furthermore, the history is not at all suggestive of carcinoma of the colon, except that the patient had had some bleeding. She could not have bled much because there was relatively no anemia.

On physical examination it is stated that the mass was tender. I am not accustomed to thinking of masses within the abdomen as being tender unless there is an inflammatory process, such as gangrene, inflammation from an ulcerated tumor or something of that sort. The normal blood chemistry, the general state of well-being, the state of nutrition and the lack of weight loss seem to indicate that this woman was in remarkably good health. Therefore, without much help from the history and little help from the physical examination, I must guess what this might have been.

If it were a mass, and from the x-ray findings, I assume it was, we are fortunate to have the roentgenologist here who did the examination. It is interesting that he was uncertain at first and repeated the examination. To me it looks like a mass producing a filling defect in the ascending colon. Although I was prepared to appear before you and say that I could see no way of making a diagnosis in this case, after seeing the x-ray films, I am willing to say that the patient probably had a benign tumor of the ascending colon. The location is too high for a lesion around the appendix, a diagnosis that we occasionally make with a tender mass in the region of the cecum. Furthermore, to me it does not look like a tuberculous lesion. Those are the conditions that sometimes confuse us in the region of the right colon. I should say that this patient had a benign tumor in the ascending colon, the exact nature of which I do not know.

FIGURE 1. *Spot Film Showing Filling Defect in Region of Ileocecal Valve.*

CLINICAL DIAGNOSIS

Polyp of ascending colon.

DR. SWEET'S DIAGNOSIS

Benign tumor of ascending colon.

Anatomical Diagnoses

Submucous lipoma of ileocecal valve.
(Chronic intussusception.)

Pathological Discussion

Dr. Castleman: I am going to ask Dr. Allen to discuss his case and then take up the pathology of both cases together.

CASE 29502

Presentation of Case

A forty-seven-year-old housewife was admitted to the hospital because of abdominal distress and vomiting of many years' duration.

During her childhood the patient was underweight and had occasional vomiting. At eighteen years of age she began to have attacks of distention, eructation, generalized abdominal pain, nausea and vomiting. The pain was most noticeable in the epigastric region and radiated through to the back and up and down the spine. The vomitus was never blood stained, nor did it contain coffee-grounds material. Vomiting occurred usually in the early evening but occasionally within five minutes of eating, especially after the ingestion of foods containing eggs and bananas. It was extreme and continued until dry retching occurred. The morning afterward she felt weak and dizzy, but there was no residual nausea. The bowel habits were regular but she was unable to describe their character. There was no change in the color of the urine. She had never been jaundiced. The attacks were not relieved by hot water or soda. She sometimes "lost as much as 12 pounds during a single attack." She had never been seen by a physician during an attack. About one month before entry she noticed severe right-sided abdominal pain, which was more prominent in the lower quadrant than in the upper and which apparently came on in the evening. "The pain felt like something would burst"; it was continuous for twenty-four hours and was associated with considerable vomiting. No medical advice was sought. It was not known whether she had had any more attacks. She remained in bed for the eighteen days before admission.

The patient was known to have had high blood pressure for many years. She had had occasional episodes of severe coughing productive of considerable sputum, which was occasionally blood tinged. Her mother was said to have had frequent "bilious attacks" with the vomiting of yellowish-green material. One sister had suffered from "stomach trouble."

Physical examination showed a slightly obese, well-developed woman in no distress. The heart was slightly enlarged, with the sounds regular and of good quality; a systolic murmur was heard in the right third interspace. The lungs were clear. The liver edge was palpable three fingerbreadths below the costal margin and was quite tender.

Figure 1. *Spot Film Showing Mass in Region of Ileocecal Valve.*

Another point of tenderness was elicited by one examiner in the right lower quadrant. There were no masses, spasm or rebound tenderness in the abdomen. Examination was otherwise not remarkable.

The blood pressure was 210 systolic, 96 diastolic. The temperature was 97.4°F., the pulse 88, and the respirations 22.

Examination of the blood showed a red-cell count of 4,500,000, with a hemoglobin of 15.6 gm. The white-cell count was 8500, with 65 per cent neutrophils. The urine was negative. A phenolsulfonephthalein test was normal. The stools were tan colored and guaiac negative. The blood nonprotein nitrogen was 19 mg. per 100 cc. A blood Hinton test was negative. A Graham test showed a 1.5-cm. area of calcification in the gall bladder, which had the appearance of a stone. The gall bladder failed to concentrate the dye.

A barium enema showed ready passage of the barium to the cecum. Arising from the lateral wall of the cecum, opposite the ileocecal valve, was a sharply defined, round mass, measuring 2 cm. in diameter (Fig. 1). The mass was constant and was well visualized after air injection. A gastrointestinal series and an intravenous pyelogram were negative.

On the fifth hospital day an abdominal exploration was performed.

Differential Diagnosis

Dr. Arthur W. Allen: May we have the x-ray demonstration at this time?

Dr. George W. Holmes: The striking thing about this case, it seems to me, is a comparison of the films taken in the usual way, after the ingestion of barium with the colon filled, and the films taken after evacuation with air in the colon. When we carry the examination a little farther and use the pressure technic and spot film we begin to see something obviously wrong in this area here. This film was taken with the cecum distended with air; this film was taken with the cecum distended with barium; and this film (Fig. 1) shows the cecum partially empty. In the text it states that the lesion was opposite the ileocecal valve. The man who did the examination had a better opportunity to determine the position than I have, but I think that the important lesion is in the region of the ileocecal valve, not opposite it. If this is correct the shadow may be due to protrusion of the ileum into the cecum. If it is opposite the valve, it must be a mass arising from the wall of the cecum. One can get all the appearances of a filling defect from a slightly edematous ileocecal valve. In the July number of the *American Journal of Roentgenology*, Golden* illustrates cases that look not unlike this that were found to be due to edema of the ileocecal valve. There is no question about this lesion in the spot films. I do not believe this is a polyp protruding from the wall.

Dr. Allen: The patient gives such a perfect story for cholelithiasis with acute inflammation and perforation into the hollow viscus, with the possibility of gallstone ileus, that I strongly suspect that there was something else the matter with her. Every time we see gallstone ileus in the ward we always say afterward that, had we been alert concerning the history of the case, we could have made the diagnosis without any question of doubt.

This woman of forty-seven had had a sensitive gastrointestinal tract since childhood. Her mother had had frequent bilious attacks, and her sister, stomach trouble. Recently I was asked to operate on the wife of a physician for gallstones. I was told that no x-ray examination had been made and the husband of the patient said: "It is not necessary. You have already operated on her mother and two of her sisters for gallstones. Besides, she has a good clinical story." This woman's early attacks were very mild and not associated with cholecystic disease. Many children vomit periodically while they are growing up, and I thought that this mass might conceivably show calcified mesenteric nodes on the x-ray films. It would have been a good sequence for her childhood disorders. At the age of eighteen, however, she began to have attacks that sound more like gallstone colic, brought on by the ingestion of foods containing a high content of fat. It is interesting that at no time during her previous life when she had these episodes or at the time of her present illness, which started a month prior to admission, had she consulted a physician. Most people who have gallstone attacks do consult a physician, usually in the middle of the night during their acute distress. Perhaps the scarcity of doctors might have played a role in her not having a physician on the night that the last acute attack started. She was apparently quite ill at that time. She was conscious of a feeling of something being radically wrong. She felt as though "something was going to burst" which, incidentally, is an interesting statement and is perhaps leading me off "on a limb." She obviously was ill because she stayed in bed eighteen days after this episode, which is about the right time for a patient with gallstone ileus to appear in a hospital, usually with vomiting and usually with a gallstone stuck about 40 cm. from the ileocecal valve, in the portion of the ileum that is narrower than the rest of the small intestine. During its descent the gallstone collects a certain amount of fecal matter on the outside, and by the time it gets to this narrow segment it may produce obstruction of the small bowel. That is the story that these patients are most apt to give. We have no history here to indicate that at any time this affair caused small-bowel obstruction. According to the story the patient certainly did not have small-bowel obstruction when she entered the hospital, nor was she extremely ill. It is a little amazing that she had a normal temperature and pulse and did not have much in the way of physical signs, except for possible tenderness in two areas — one in the region of the right upper quadrant and one in the region of the right lower quadrant. So she could, I think, have had a gallstone that had left the gall bladder, had entered the gastrointestinal tract and was discovered

*Golden, R. Enlargement of ileocecal valve. *Am. J. Roentgenol.* 50:19-23, 1943.

in the region of the cecum at the time of admission.

There are a great many other things that might explain the local picture in this region. This woman had had no history of bleeding from the bowel. She was free from anemia — as a matter of fact she had 15.6 gm. of hemoglobin at entry, which is above normal. That brings up the question of tumors of the right bowel, which are supposed to cause anemia but which, as a matter of fact, only cause anemia in about 70 per cent of the cases. This could perfectly well have been a benign tumor, such as a lipoma, a lipofibroma or a true fibromyoma, or a benign polypoid lesion, although I strongly suspect that it was not of that type because she came to the hospital following an acute episode. This type of tumor does not cause an acute episode, except for intussusception, which does not permit a patient to stay in bed eighteen days prior to admission to the hospital; but an inflammatory lesion such as appendicitis could. That also could produce a defect similar to the one that we see here, but if the defect were still present from such cause I should expect that there would still be some leukocytic elevation, some elevation of temperature and some obvious tenderness in this region. Besides, in this film one sees a perfectly normal appendix. So I shall rule out appendicitis. Occasionally a single diverticulum of the intestine with diverticulitis occurs in this site. These cases produce symptoms and signs of acute appendicitis and are always diagnosed acute appendicitis preoperatively. The history is usually shorter than this and associated with a perfectly typical febrile reaction and a tender inflammatory mass. Therefore, I shall rule that out.

I am going to go back to the gallstones in this case: the history is so perfect for gallstone ileus, and about the right time had elapsed after the severe onset. One gallstone was left behind, and although the mass in the cecum does not look like a gallstone to me, I am going to assume that there was a gallstone in the cecum at the time of operation.

Dr. Holmes: The patient had a barium meal, and it went through without difficulty.

Dr. Allen: I should expect a barium meal to show the biliary system if she had a fistula. Did you purposely keep back the gall-bladder films?

Dr. Holmes: No. I did not put them up because they do not show anything. If the patient had gallstone ileus, she should have shown some obstruction.

Dr. Allen: Yes; I should assume there was no small bowel obstruction from the appearance of the flat film. No loops of small bowel are apparent.

Dr. Holmes: I should have put up the other film. I neglected to do so because it did not show anything. But you notice the barium passed through the ileum all right and entered the cecum.

Dr. Allen: That would be expected if the gallstone had gone by, but I should like to see a little barium in the biliary tree.

Clinical Diagnoses

Cecal polyp.
Chronic cholecystitis and cholelithiasis.

Dr. Allen's Diagnoses

Gallstone in cecum.
Cholelithiasis.

Anatomical Diagnoses

Hypertrophied ileocecal valve.
(Chronic intussusception.)
Cholelithiasis.

Pathological Discussion

Dr. Castleman: In the case that Dr. Allen discussed the patient was operated on and the surgeon found the gall bladder filled with stones. There was no connection that he could make out between the gall bladder and the duodenum or the jejunum. When he felt the cecum he was able to palpate a definite mass. On opening the cecum he found a prolapse or protrusion of the mucosa (and possibly of the entire wall) of the terminal ileum into the cecum for a distance of 2.5 to 3.5 cm. The edge of the protruding tissue was everted. The whole mass of tissue resembled the end of a cornet horn. The examining finger passed freely through the lumen of the protruding tissue into the terminal ileum. No obstruction was felt. It was thought that any procedure toward removal of the lesion would necessitate an ileotransverse colostomy and right colectomy in a two-stage operation. Since the patient had hypertensive heart disease and since there was no obstruction in the ileocecal region no further surgery was deemed advisable at that time.

The patient left the hospital and was apparently free from symptoms for over two years, when she died of a cerebral hemorrhage.

In the case discussed by Dr. Sweet, the surgeon did not open the cecum. He felt the mass and resected the cecum and the terminal ileum. When the specimen was opened, the mass was found to be an ovoid, well-circumscribed, submucosal tumor involving about a third of the circumference of the ileocecal valve and projecting about 0.7 cm. above the rest of the valve. The mucosa over the lesion was definitely hemorrhagic. When sectioned the mass proved to be a lipoma. In

the serosa over both ileum and cecum there were many areas of fat necrosis. This, together with the mucosal inflammation, is evidence that something had been going on, and certainly the likeliest process is chronic intussusception.

The problem in Dr. Sweet's case is to decide whether the mass was a true lipoma or merely a hypertrophied ileocecal valve, with perhaps more than the usual amount of submucosal fat, similar to the one found in Dr. Allen's case. In going through some of our routine autopsies we have found hypertrophied ileocecal valves that had been removed from patients who had had no symptoms of chronic intussusception. These ileocecal valves are fatty and uniformly enlarged, unlike that in Dr. Sweet's case, in which the lesion was circumscribed and had produced a localized elevation of the mucosa. The case discussed by Dr. Sweet I think should be called a true lipoma, although I admit that I am not sure it could not have been merely a variation from the normal ileocecal valve. We have seen several other obvious submucosal lipomas in the intestinal tract, especially in the cecum and ascending colon. These were much larger than the one under discussion today and had produced intussusception. I believe that in the case discussed by Dr. Allen the patient had a hypertrophied ileocecal valve that also produced chronic intussusception.

Dr. Allen: I should like to ask Dr. Holmes if he considers the case I discussed as typical of the cases Golden described. I think he spoke of mucosal edema and not of fat deposits.

Dr. Holmes: That is correct.

Dr. Allen: It seems to me that these cases represent either an unusually large ileocecal valve or a small herniation of the terminal ileum through the valve. Is it not true that we may see a considerable variation? We have no good idea of what they are normally. How do you reconstruct the acute episode starting one month prior to entry?

Dr. Castleman: The patient probably was not sick because of her ileocecal intussusception. She was a severe cardiac and undoubtedly went to bed for any mild complaint. I believe that she had intussusception off and on, which reduced itself spontaneously. She remained free from symptoms for almost two years after operation.

Dr. Holmes: The radiologist is more or less responsible for getting the surgeons into difficulties, and we must eventually be able to recognize this condition and to distinguish it from true tumor of the cecum.

Dr. Castleman: Does it matter whether you call it a true tumor or a variation from the normal? If it produces chronic intussusception, perhaps something should be done surgically.

Dr. Holmes: The points are that the surgeon might not be in so much of a hurry to operate and that he would not take the cecum out if it were due to protrusion of the valve.

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A CHRISTMAS MESSAGE

With our third wartime Christmas at hand we can, at last, look back on an event, and gradually appreciate its meaning, that will have an impact on the whole Christian world. The three wise men have met in the shadow of the ageless pyramids, one from the North, one from the East, and one from the West, and have solved the riddle of the Sphinx — that without unity there can be no strength.

The wise men departed, two of them to meet again still farther to the east, with a fourth wise man, and the same principle was reaffirmed — that only separately can the sticks of the bundle be broken. Thus, can the lessons taught even by the Fascisti be reapplied and made use of in a righteous cause.

Despite the havoc and the carnage and the increasing tempo of battle, and the dreadful seeds of destruction that are being sown with such merciless patience on the modern cities of Babylon, we can at last see the pattern of the fabric we are weaving. With the promise of unified efforts, now and hereafter, we can begin to visualize — and it gives us hope for this season — a future of peace on earth to men of goodwill.

We have never made the battlefield the shrine of our national devotion; creators by instinct and practice, we can take only a passive and somewhat horrified pride in the perfection of our skill at destruction; yet we cannot sicken of the battle until those bent on our own destruction have sickened utterly of it.

As a Christmas, this cannot be a merry one. There is too much sadness, too much anxiety in our hearts. It can, however, be a Christmas of fierce and militant joy and of increasing courage, for despite our sorrows and because of our sacrifices, Christ will be reborn, and peace will once more return.

PUBLIC HEALTH IN BOSTON

An eighty-page booklet, *Boston's Health in 1942*, has recently been issued as Document 13 by the Printing Department of the City of Boston. It is the seventy-first annual report of the Boston Health Department, and is in every way worthy of its deluxe printing.

Among the more interesting items is a chart of the recorded deaths in Boston since 1725. The gross death rates for 1941 and 1942 (12.5 per 1000 population) are the lowest ever recorded, and come at the end of two decades in which the rates were consistently under 16, a figure that was reached only once in the preceding two centuries. The notable peaks were 28 in 1821 (measles); 38 in 1849 (cholera), 31 in 1872 (smallpox) and 23 in 1918 (influenza). The low rates of the past quarter century are due to the improved sanitary standards of modern times, undoubtedly in combination with a greater resistance to infectious

diseases on the part of a better nourished population.

To the medical profession much of the information in this report merely confirms what it well knows—that Boston continues to have an ably administered Health Department. A greater value, and one that the *Journal* hails, is that the report will be read in circles not usually reached by medical publications, and will take information on public health to places where it would not otherwise have been carried.

MEDICAL EPONYM

Neufeld Method

A note on the observation of specific capsular swelling of pneumococci by Ferdinand Neufeld (b. 1869) first appeared in *Zeitschrift für Hygiene und Infectionskrankheiten* (**40:** 54–72, 1902) under the title "Über die Agglutination der Pneumokokken und über die Theorieen der Agglutination [Agglutination of Pneumococci, and Theories of Agglutination]." A portion of the translation from page 57 follows:

> If we mix equal parts of agglutinating serum and a bouillon culture of pneumococci, whether in the test tube or by intimate admixture on a coverslip of a loopful each of serum and culture, we observe in the hanging drop preparation distinct signs of swelling, which in the case of a strongly potent serum appear immediately or after only a few minutes. The individual cocci swell up twice or three times their [normal] size, and at the same time flatten out at their points of contact with each other, which are ordinarily pointed, and their whole contour becomes indistinct and hazy.

Twenty-nine years later, in an article with R. Etinger-Tulczynska entitled "Nasale Pneumokokkeninfektionen und Pneumokokkenkeimträger im Tierversuch [Nasal Pneumococcal Infections and Pneumococcus Carriers in Animal Experiments]," which appeared in the same journal (**112:** 492–526, 1931), Professor Neufeld describes the inoculation of bouillon with nasal secretion. A portion of the translation from page 495 follows:

> The serum bouillon growth usually does not show a pure culture of pneumococci, but a mixture of various bacteria (among them, frequently, streptococci). Notwithstanding, it is possible quickly and with certainty to identify typical pneumococci if advantage is taken of the fact that they promptly swell markedly in homologous rabbit (not horse!) serum. . . .

And in a footnote on page 496 he observes:

> The swelling phenomenon has furthermore proved its worth to us as a simpler and more reliable means of typing pneumococci than the usual agglutination method.

R. W. B.

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

THE PHYSICIAN'S ROLE IN TUBERCULOSIS CASE-FINDING IN INDUSTRY

Some Massachusetts physicians have received a letter from the Division of Tuberculosis stating that significant findings were noted in an x-ray film of the doctor's patient during an industrial survey of the plant where the patient is employed. A report of the x-ray findings is given, together with the information that the employee has been notified to consult his family physician so they may talk over the results of the report.

Routine x-ray examination of the chest for the purpose of finding unsuspected pulmonary tuberculosis in apparently healthy people is not only being used in the induction centers for the Army and Navy, but also in pre-employment examination in large industries and in the case-finding programs of state health departments and the United States Public Health Service.

Efficiency of the x-ray. There is no question that the x-ray method is the most effective single means for finding unsuspected pulmonary tuberculosis. Its value is limited only by the extent to which it is used. A person may have pulmonary tuberculosis in any stage of the disease either without any symptoms or with symptoms that are not alarming enough to cause him to seek medical advice. In early cases of tuberculosis, the x-ray evidence may be present three to five years before the disease has progressed to the symptomatic stage.

The absence of symptoms does not mean that the disease is not in an infective stage. In a recent study of 160 Army rejectees for pulmonary tuberculosis at the Middlesex County Sanatorium, it was found that only 10 of these had been previously known to have tuberculosis, and all were classed 1A by their local draft boards. By stage of disease, these cases were 75 per cent minimal, 14 per cent moderately advanced and 11 per cent advanced. The most significant fact from the public-health point of view is that over half the patients with moderately advanced and advanced cases had positive sputums, and even 8 per cent of those with minimal cases had positive sputums. Such unsuspected bacillary cases are continuously seeding their communities with tuberculosis, and must be found and segregated if tuberculosis is to be eliminated or reduced to a low level.

New program of case-finding. In order to pick up these undiagnosed cases, the Massachusetts De-

partment of Public Health, in co-operation with the Massachusetts Tuberculosis League, inaugurated a program of pulmonary tuberculosis case-finding in industry on September 28, 1943. His Excellency, Governor Leverett Saltonstall, was the first to have an x-ray film of his chest, and he was followed by the State House employees. The x-raying is accomplished by means of a fully equipped mobile unit which takes a 4-by-10-inch stereoscopic photofluorogram at a cost of only 6 cents per film. The unit was purchased by the Christmas-Seal-supported tuberculosis associations of Massachusetts and is loaned to the department. X-ray surveys are conducted in industry on a voluntary basis and at the invitation of the plant management.

Follow-up of suspects. On the identification blanks that are being used, the employee is asked to name the physician to whom he wishes his x-ray report to be sent. If the x-ray findings are significant, a written report is sent to the physician and the employee notified by mail to see his doctor. It is hoped that when the latter presents himself to his physician for interpretation of the x-ray findings, he will be placed under careful clinical, laboratory and further x-ray studies to confirm or deny the impression gained from the first roentgenogram. If the diagnosis of pulmonary tuberculosis is confirmed, the physician should then report the case to the local board of health.

It is the responsibility of the district health officer to see that the employee found to have suspected tuberculosis presents himself to his physician for further studies and, if the diagnosis is confirmed, that the case is reported to the local board of health in the prescribed manner.

COMMUNICABLE DISEASES IN MASSACHUSETTS FOR NOVEMBER, 1943

Résumé

Diseases	November 1943	November 1942	Seven-Year Median
Anterior poliomyelitis	25	2	4
Chicken pox	1282	969	969
Diphtheria	31	5	14
Dog bite	600	564	598
Dysentery, bacillary	22	30	30
German measles	64	61	39
Gonorrhea	510	378	421
Measles	816	1073	597
Meningitis, meningococcal	44	12	8
Meningitis, other forms	9	6	*
Meningitis, undetermined	7	1	*
Mumps	398	639	358
Pneumonia, lobar	224	233	269
Salmonella infections	7	7	5
Scarlet fever	707	882	493
Syphilis	617	535	469
Tuberculosis, pulmonary	247	220	220
Tuberculosis, other forms	22	17	23
Typhoid fever	3	0	6
Undulant fever	6	0	5
Whooping cough	407	952	710

*Pfeiffer-bacillus meningitis only other form reportable previous to 1941.

Comment

Anterior poliomyelitis, as might be expected, is now definitely on the downward grade. The meningococcal meningitis figure has remained in the forties for the last four months. This is a decidedly high level compared with that of other years. Diphtheria has been creeping up since August; the November figure is the highest for this month since 1935, but this year's total probably will not exceed that of last year. Whooping cough, on the other hand, shows a 50 per cent decline over last November.

Geographical Distribution of Certain Diseases

Actinomycosis was reported from: Springfield, 1; total, 1.

Anterior poliomyelitis was reported from: Adams, 2; Beverly, 2; Boston, 4; Cambridge, 1; Danvers, 1; Fall River, 3; Haverhill, 1; Lynn, 3; Natick, 1; New Bedford, 1; North Adams, 1; Norwood, 1; Pittsfield, 1; Saugus, 1; Seekonk, 1; Watertown, 1; total, 25.

Diphtheria was reported from: Boston, 12; Cambridge, 1; Chelsea, 1; Foxboro, 1; Gloucester, 1; Lowell, 4; New Bedford, 3; Salem, 1; Somerville, 4; Woburn, 3; total, 31.

Dysentery, bacillary, was reported from: Boston, 6; Brookline, 1; Cambridge, 2; Canton, 1; Hingham, 1; Ipswich, 1; Lowell, 4; Malden, 2; Melrose, 1; Quincy, 1; Stoneham, 1; Wellesley, 1; total, 22.

Encephalitis, infectious, was reported from: Braintree, 1; Brockton, 1; Fort Devens, 1; total, 3.

Malaria was reported from: Falmouth, 1; Fort Banks, 13; Fort Devens, 11; total, 25.

Meningitis, meningococcal, was reported from: Agawam, 1; Boston, 7; Brockton, 1; Cambridge, 1; Fall River, 5; Fitchburg, 2; Fort Banks, 1; Fort Devens, 1; Haverhill, 1; Holbrook, 1; Holden, 1; Leominster, 1; Lowell, 1; Lynn, 2; New Bedford, 1; Norwood, 1; Revere, 1; Saugus, 1; Somerville, 3; Southbridge, 1; Springfield, 2; Waltham, 1; Weymouth, 1; Worcester, 6; total, 44.

Meningitis, other forms, was reported from: Boston, 1; Holyoke, 1; Lawrence, 1; Malden, 1; Medford, 1; Milford, 1; Somerville, 1; Springfield, 1; Worcester, 1; total, 9.

Meningitis, undetermined, was reported from: Boston, 3; Cambridge, 3; Leominster, 1; total, 7.

Salmonella infections were reported from: Boston, 1; Cambridge, 2; Marblehead, 2; Wellesley, 2; total, 7.

Septic sore throat was reported from: Boston, 6; Clinton, 1; Malden, 1; Marion, 2; Milton, 1; New Bedford, 1; total, 12.

Trichinosis was reported from: Newton, 1; total, 1.

Typhoid fever was reported from: Lowell, 1; Malden, 1; North Attleboro, 1; total, 3.

Typhus fever was reported from: Williamstown, 1; total, 1.

Undulant fever was reported from: Dartmouth, 1; Easton, 2; Framingham, 1; Somerville, 1; Wareham, 1; total, 6.

CONSULTATION CLINICS FOR CRIPPLED CHILDREN IN MASSACHUSETTS UNDER THE PROVISIONS OF THE SOCIAL SECURITY ACT

Clinic	Date	Clinic Consultant
Salem	January 3	Paul W. Hugenberger
Haverhill	January 5	William T. Green
Lowell	January 7	Albert H. Brewster
Brockton	January 13	George W. Van Gorder
Pittsfield	January 17	Frank A. Slowick
Springfield	January 19	Garry deN. Hough, Jr.
Worcester	January 21	John W. O'Meara
Fall River	January 24	Eugene A. McCarthy
Hyannis	January 25	Paul L. Norton

WAR ACTIVITIES

CIVILIAN DEFENSE

OCD Affiliated Units

The United States Office of Civilian Defense has recently announced that ninety-three hospitals and medical schools scattered throughout the country have completed the formation of affiliated units of civilian physicians, which will be available to either OCD or the Army in the event of need for setting up emergency hospital facilities in their respective areas.

Each unit is composed of fifteen physicians, surgeons and other specialists, and forms a balanced professional staff. OCD will use the units to supplement the staffs of emergency base hospitals located in relatively safe zones on the fringes of critical areas in case it is necessary to transfer civilian patients to these hospitals because of emergency in such areas. Furthermore, the units will be called on by the War Department to staff extemporized hospitals should there be a sudden influx of battle-front casualties, or some other extraordinary military necessity, requiring hospitals and physicians beyond the immediate capacity of the Army in any particular locality. The affiliated units will be used for military emergency purposes only in or near the communities in which the staff resides. Their duties will be temporary and they will be replaced by Army medical officers as quickly as the Surgeon General of the Army can make the necessary assignments.

Normally, the fifteen doctors of a unit are associated with a single hospital. Each unit includes a chief and assistant chief of medical services, two general internists, a chief and assistant chief of surgical services, four general surgeons, two orthopedic surgeons, one dental surgeon, one pathologist and one radiologist. Physicians accepted for service in the units receive inactive reserve commissions in the United States Public Health Service, but will be called to active duty by the Surgeon General (USPHS) only at the request of OCD. When a unit is needed, either to staff an emergency base hospital or to assist the Army temporarily in a military emergency, the physicians of the unit will be placed on active duty for the duration of that particular emergency.

Following is a list of units completed and commissioned by the Public Health Service up to October 30, 1943, in Region I, which includes all the New England States:

Boston University School of Medicine, Boston
Cambridge Hospital, Cambridge, Mass.
Goddard Hospital, Brockton, Mass.
Harvard Medical School (A), Boston
St. Luke's Hospital, Pittsfield, Mass.
Springfield Hospital, Springfield, Mass.
Central Maine General Hospital, Lewiston, Maine
Eastern Maine General Hospital, Bangor, Maine
Maine General Hospital, Portland, Maine
Elliot Hospital, Manchester, New Hampshire
Lawrence and Memorial Associated Hospitals, New London, Conn.
Meriden Hospital, Meriden, Conn.
Stamford Hospital, Stamford, Conn.
Waterbury Hospital, Waterbury, Conn.
Yale University School of Medicine, New Haven, Conn.
Rhode Island Hospital, Providence, Rhode Island
St. Joseph's Hospital, Providence, Rhode Island

MISCELLANY

CANCER OF THE LUNG— A GROWING PROBLEM

Until recently, primary carcinoma of the lung was regarded as a relatively rare type of cancer. We are now recognizing that it is one of the commonest forms of neoplasm. It has been discovered that around 10 per cent of all cancers originate in the lung, that this organ is surpassed only by the stomach as the most frequent site of beginning malignancy, and that approximately 15,000 Americans succumb annually to carcinomas that arise from lung structures, usually in the bronchi. Such stark statistics and the demonstrated fact that cancer of the lung, like pulmonary tuberculosis, can be found early by employment of readily available diagnostic facilities, provide all physicians with food for thought as they evaluate the chest complaints presented by patients, especially men, and particularly those past forty years of age. Many of the pertinent points are discussed in a recent paper (Overholt, R. H. A common masquerading lung disease. *Dis. of Chest* 9:197–210, 1943).

There is a masquerading lung disease which often gives quarter for a short time before the fatal issue and whose actions, in many ways, may simulate those of tuberculosis.

Both diseases are unique for they masquerade as other acute or chronic conditions of the lung. In neither are symptoms reliable in the early stages. Both diseases are marked by a lack of early reliable physical signs. Both are unique since in the early stages a single x-ray film will usually show some abnormality. Again, they ape one another because in spite of obscure clinical factors the diagnosis can be accurately made in a high percentage of cases. Lastly, there is a similarity between tuberculosis and this masquerading disease, cancer of the lung, as successful treatment depends to such a large degree on early discovery.

However, the two diseases are different as regards the predominant age groups affected. Tuberculosis concerns principally the age groups between fifteen and forty, whereas lung cancer usually affects those between the ages of forty and sixty-five. The diseases are totally different in respect to the matter of time. In tuberculosis, time plus rest is often a useful ally of the patient in regaining health. In cancer of the lung the element of time is always an enemy of the patient. Prolonged observation and rest treatment never improve the situation, but rob the patient of his only chance for possible cure.

In 165 cases of lung cancer it was found that the patients first consulted a doctor because of symptoms usually associated with almost any chronic chest condition. A review of these symptoms suggests it would be impossible to set apart any group of complaints that could be regarded as pathognomonic of pulmonary malignancy. Nevertheless, 82 per cent of all the patients reported chronic cough, whereas no less than 92 per cent had as a first symptom something that called for attention to be directed to the chest when first the physician was consulted. Besides cough, other common symptoms included chest pain, chills and fever, hemoptysis, dyspnea, loss of weight and weakness.

Reviewing the physical signs elicited it is again impossible to outline a specific and significant grouping any more suggestive of cancer than of other chronic pulmonary conditions. Cases examined in the early stages often presented no physical signs. When present, the signs were of considerable variety and frequently misleading. They included evidence of congestion, consolidation, fluid, localized emphysema, cavitation, bronchial obstruction, mediastinal shift and other phenomena varying with the case, thus emphasizing the unreliability of simple physical signs in the differential diagnosis of this condition.

Of the 165 cases, 104 (63 per cent) were incorrectly diagnosed by the first doctor consulted. In view of the confused picture of misleading symptoms and physical findings, perhaps this majority in favor of error is not completely surprising, but the sobering thought emerges that treatment based on an erroneous diagnosis was maintained for long periods of time, aimed at such supposed conditions as tuberculosis, 40 cases; unresolved pneumonia, 18 cases; lung abscess, 13 cases; bronchitis, 11 cases; asthma, 5; heart disease, 4; pleurisy, 4; metastatic tumors, 2; and miscellaneous, 9 cases. The most notable fact was the high frequency of false diagnoses of tuberculosis.

Unfortunately, lung cancer was not unmasked in far too many cases until long after the patient first visited a physician. It was possible in the case histories of 125 patients to determine how speedily a verified diagnosis was reached. Two facts stood out boldly. First, 36 per cent of the patients placed themselves under medical supervision at onset or within one month of the onset of symptoms. Second, the average patient consulted a doctor within three months of onset but did not receive benefit of a chest x-ray film for an additional three months. The true diagnosis was not arrived at until nine months had elapsed from the time when the first doctor saw the patient.

The x-ray, without doubt, is by far the most valuable aid in apprehending pulmonary disease, but a distinction is necessary between its ability to yield presumptive and absolute evidence. In 98 per cent of this series of cases the initial film revealed that trouble was present. An explanation of the delay in reaching a final diagnosis may be found in the fact that in the majority of cases the primary pathologic process failed to produce on the film or the fluoroscopic screen a shadow of itself. Those abnormalities that did appear were secondary effects due to the presence of the neoplasm and were of such variability as to be susceptible of a wide range of interpretation.

In 95 per cent of the cases it was possible to establish an unequivocal diagnosis during life, bronchoscopy being the leading method of obtaining tissue, and having been employed in 103 cases. In 39 other cases surgical exploration was used. Metastases were sectioned in a few cases, aspiration was the method in another small group, whereas the remaining 5 per cent were diagnosed only after postmortem examination.

For a decade, surgery has been available in the treatment of lung cancer. A creditable showing has been made during this pioneering period. For example, 2 out of every 5 cases surgically explored have been found to be free of extension of the cancer extrapulmonarily. In 20 per cent of the entire group of verified cases there was some hope of cure. This seems an encouraging ratio when we recall that prior to 1933 there was no reason to regard the condition as anything but incurable. As a reward for our efforts, 20 patients, or 13 per cent, remain as the net salvage from the entire series of 156 verified cases of primary lung cancer, out of 32 selected for an attempt at curative resection. These 20 patients are all reasonably well and devoid of evidence of metastatic disease, and 5 of them can be referred to as "cures" in so far as they have now passed the five-year mark.

In considering practical steps toward bringing cases of lung cancer to light during the curable stage we can learn valuable lessons from the record on tuberculosis case finding. Physicians have been taught that if tuberculosis is to be discovered during its minimal stage it is not necessary to search for absent or insignificant symptoms and physical signs but to go immediately to the x-ray. The same can be said for the apprehension of early lung cancer.

How may the first doctor consulted set in motion this mechanism of early discovery? He may save valuable time for his patient if he remembers:

1. That cancer of the lung is now one of the most important diseases of the chest in patients within the age period from forty to sixty-five years, particularly in males.
2. That many patients do seek help at a time when the lesion is still confined to the lung.
3. That symptoms and signs are either lacking or misleading in the early stages.
4. That the earliest lesions will in almost every case produce some telltale shadow on the x-ray film, and
5. Finally, that there are two methods available for clinching the diagnosis: first, that the majority of lesions are visible bronchoscopically and accessible for biopsy, and second, that when the suspicion cannot be verified in this way, it is possible to explore the chest safely by surgical means, settle the diagnosis and carry out curative treatment if necessary.

— Reprinted from *Tuberculosis Abstracts* (December, 1943).

NOTICES

BOSTON COUNCIL OF SOCIAL AGENCIES

The following series of lectures covering the problem of alcoholism from various aspects will be given at Zero Marlborough Street, Boston, on Friday evenings at 7:30 p.m. They are sponsored by the Committee on Alcoholism of the Boston Council of Social Agencies.

January 7 — The Heredity of the Inebriate. Dr. E. M. Jellinek, associate professor of applied physiology, Yale University.

February 11 — Alcohol and Public Opinion. Dr. Dwight Anderson, director, public relations, Medical Society of the State of New York.

March 10 — Effects of Alcohol: The evidence of physiological experiments. Dr. Howard W. Haggard, director, Laboratory of Applied Physiology, Yale University.

April 7 — The Treatment of Chronic Alcoholism. Dr. Robert Fleming.
Plan of Treatment at the Washingtonian Hospital with Special Consideration of the Conditioned Reflex Treatment. Dr. Joseph Thimann.

May 12 — Alcoholism, Criminology, and Parole. Reuben L. Lurie, chairman, parole board, Massachusetts Department of Correction.

These lectures will be open to the public.

(Continued on page xii)

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To Betty it's all that a brand-new permanent can mean to a woman. Something to fortify the spirit . . . to do the soul good.

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A glass of beer—a small thing, surely—not of crucial importance to any of us. And yet—morale *is* a lot of little things like this.

Little things that help to lift the spirit, keep up the courage. Little things that are part and parcel of our own American way of life.

And, after all, aren't they among the things we fight for?

MORALE IS A LOT OF LITTLE THINGS

(as you, Doctor, know better than most)

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ANNOUNCEMENT

Dr. John L. Newell announces the reopening of his office for the practice of obstetrics and gynecology at 330 Dartmouth Street, Boston.

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Medical Conference Program

Wednesday, January 5 — The Adrenal Gland in Relation to Neuromuscular Fatigue: (Studies on Pilots). Dr. Nicholas T. Werthessen.

Friday, January 7 — Hypoestrinism. Dr. Fuller Albright.

Wednesday, January 12 — Sinusitis and Obesity. Dr. George Kelemen.

Friday, January 14 — Neurosurgical Problems in Early Life. Dr. Franc D. Ingraham.

Wednesday, January 19 — Atopic Eczema. Dr. Ethan Allan Brown.

Friday, January 21 — The Present Status of the Etiology of Hypertension. Dr. Lewis Dexter.

Wednesday, January 26 — Urological Problems in Infants and Children. Dr. James Marvin Baty.

Friday, January 28 — Clinical Experiences with Purified Human Serum Albumin. Dr. Charles A. Janeway.

On Tuesday and Thursday mornings Dr. S. J. Thannhauser will give medical clinics on hospital cases.

NEW ENGLAND HOSPITAL
FOR WOMEN AND CHILDREN

The monthly clinical conference and meeting of the staff of the New England Hospital for Women and Children will be held on Thursday, January 6, at 7:15 p.m. in the classroom of the nurses' residence. Lt. John L. Ward (MC), U.S.N.R., will speak on the subject, "Venereal Disease Program of the Navy in Its Relation to Public Health." Dr. Winnifred P. Chase will be chairman.

AMERICAN BOARD OF OPHTHALMOLOGY

The American Board of Ophthalmology announces the following examinations: New York City, June 3 and 4 — deadline, December 15, 1943; and Chicago, October 5, 6 and 7 — deadline, April 1, 1944. All applications received after January 1, 1944, will be subject to the increased total fee of $75.00.

Effective January 1, 1944, the address of the Executive Office will be P. O. Box 1940, Portland 2, Maine, to which all official correspondence should be forwarded.

CHILDREN'S HOSPITAL

Thursday, January 6 — Experiences with Esophageal Atresia and Tracheo-esophageal Fistulas. Dr. William E. Ladd.

Thursday, January 13 — Mechanisms Concerned in Some Hemolytic Anemias. Dr. William B. Castle.

Thursday, January 20 — Problems in the Treatment of Infantile Paralysis. Dr. William T. Green.

Thursday, January 27 — The Roentgen Diagnosis of Bone Tumors. Dr. Albert B. Ferguson.

These meetings are scheduled to begin at 5 o'clock and are open to the public.

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*A Simple Micromethod for the Detection of Acetone in Urine. Brem, J., *New Eng. J. Med.*, 224:462, 1941.

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EDWARD K. DUNHAM LECTURES

The Faculty of Medicine of Harvard University has announced that the following lectures on the general topic, "Some Virus Diseases of Man: Evolutionary and ecologic considerations" will be delivered by Dr. Frank Macfarlane Burnet, under the Edward K. Dunham Lectureship for the Promotion of Medical Sciences:

Monday, January 10. The Origins of Human Infectious Disease.
Wednesday, January 12. Herpes and Poliomyelitis.
Friday, January 14. Influenza.

These lectures are scheduled for 5 p.m. in the amphitheater of Building C, Harvard Medical School.

SOCIETY MEETINGS AND CONFERENCES

Calendar of Boston District for the Week Beginning Thursday, December 23

January 5–28. Joseph H. Pratt Diagnostic Hospital. Medical conference program. Page xii.
January 6. New England Hospital for Women and Children. Page xii.
January 6–27. Children's Hospital, Boston. Page xii.
January 7-May 12. Boston Council of Social Agencies. Page 958.
January 10, 12 and 14. Edward K. Dunham Lectures. Notice above.
January 13. Sudden Death. Dr. Alan R. Moritz. Pentucket Association of Physicians. 8:30 p.m. Haverhill.
February 12. American Board of Obstetrics and Gynecology. Page ix, issue of October 28.
May 23 and 24. Massachusetts Medical Society. Annual meeting, Hotel Statler, Boston.
June 12–16. American Medical Association. Annual meeting, Chicago. Page 798, issue of November 18.

District Medical Society

WORCESTER
January 12. Memorial Hospital, Worcester.
February 9. Worcester State Hospital, Worcester.
March 8. St. Vincent Hospital, Worcester.
April 12. Hahnemann Hospital, Worcester.
May 10. Annual meeting.

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